Cardiac Auscultation

INCLUDING AUDIO VISUAL PRINCIPLES

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FOREWORD

TEACHING has been the primary persistent interest of our group the emphasis being on knowledge that the clinician could use in his own practice The techniques of teaching auscultation have always been difficult, but prevailing were two serious obstacles (1) The almost intolerable boredom of the student and instructor while waiting for others to listen (2) The reluctance of students to time sounds and murmurs These two faults have been greatly minimized by the development and use of the audiovisual methods

Auscultation comprises only a part of the evaluation of cardiac status but it is important because doctors despite refinement of a multitude of other diagnostic methods will continue to listen And the listening will be critical Ward rounds without some controversy about a murmur are unusual However because of the objective accuracy of present day methods empiricism no longer rules

The authors deserve thanks for presenting a stimulating guide toward understanding the problems of listening to the heart The credit is theirs particularly J S B s whose years of work on the development and use of the method herein described have resulted in a significant advance in teaching

To the Melville family whose generosity provided funds for J S B and M C when funds were not so easily obtained and to Charles McLeod who gave the first few hundred dollars for the development of the cardioscope go my added appreciation now as then The donors claimed no scientific knowledge but had the innate sense of the successful namely preferring to invest in individuals *rather than in a problem a principle that can profitably* have wider application

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I hope the book is well received and that it will emphasize to its readers that auscultation can be of great help when understood and evaluated with other findings

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March 1955

PREFACE

THIS BOOK is the outgrowth of a course in auscultation of the heart given for the past few years at the New York University Post Graduate Medical School. The purpose of both the book and the course is to teach clinical auscultation. Audio-visual techniques developed over a period of years have been used in this course and it has been observed that the simultaneous delivery of sound to the ear and of the visual pattern of the sound to the eye has great advantages over either alone both for instruction and for decision in difficult problems of auscultation. Equipment for this purpose is described and illustrated.

The book emphasizes the importance of auscultation in examination of the heart and contains over fifty illustrations of which the majority are stethograms, the others being concerned with basic principles. Many theoretical considerations and controversial points have been omitted with the intention of keeping the book both short and practical.

It is a pleasure to thank the Cambridge Instrument Company, Dr. C. A. Poindexter and members of the Division of Cardiology of the New York University Post Graduate Medical School for help and cooperation, and Dr. Edmund H. Reppert for his suggestions and criticisms. The excellent drawings and charts are the work of Olive Emslie and we are most grateful for her help. Finally, three uncomplaining ladies who have tolerated much receive our thanks.

10 March 1955

J. S. B.
M. R. C.
R. McG.

This book is affectionately dedicated to DR ROBERT HURTIN HALSEY *eminent cardiologist founder in 1908 of the cardiac clinic of the New York Post Graduate Medical School and Hospital and one of the leaders in the organization of the New York Heart Association in 1914 and the American Heart Association in 1922*

CHAPTER I

Historical Aspects of Auscultation of the Heart

WILLIAM HARVEY is generally regarded as the discoverer of the circulation of the blood His book An Anatomical Dissertation Upon the Movement of the Heart and Blood in Animals [1] was published in Frankfurt in 1628 This work contains the complete physiology of the circulation with the exception of the capillaries which Harvey foretold and which the invention of the microscope allowed Malpighi to discover in the mesentery of the frog in 1661 It is often said of Harvey that he failed to give due credit to Servetus Columbo and Cesalpino who held certain basic ideas regarding the circulation but in his own words he began his studies to learn from actual inspection and not from the writings of others Perhaps it should be said of Harvey that he demonstrated the circulation rather than that he discovered it and about this there can be no disagreement

In his book Harvey mentions the heart sounds saying that with each movement of the heart when there is the delivery of a quantity of blood from the veins to the arteries a pulse takes place and can be heard within the chest [2] Inasmuch as immediate or direct auscultation in which the ear is placed directly against the chest has been known since the time of Hippocrates it seems certain that heart sounds had been heard previous to Harvey's observation but the interpretation placed upon these sounds is unknown

It appears however that the knowledge of the existence of heart sounds and the use of immediate auscultation had added nothing of material value regarding the heart and its diseases This

came with publication of the book by Rene Theophile Hyacinthe Laennec appearing in France in 1819 under the title of "A Treatise on Mediate Auscultation"[4] which introduced mediate or indirect auscultation by means of the stethoscope (Gr *stethos,* breast + *skopos* watcher)

It was Laennec's opinion that the heart sounds were unrelated to the impulse of the heart against the chest wall He stated that the first heart sound resulted from ventricular contraction and the second sound from auricular contraction This explanation proved to be incorrect as did certain of his ideas regarding heart murmurs However Laennec gave the first careful descriptions of the auscultatory findings of the normal heart and of many varieties of heart disease and he is regarded as the founder of the art of auscultation of both the heart and the lungs

At the time Laennec discovered the principle of mediate auscultation he was physician to the Necker Hospital in Paris His own words best describe the discovery "In 1816 I was consulted by a young woman labouring under the general symptoms of diseased heart and in whose case percussion and the application of the hand were of little avail on account of the great degree of fatness The other method just mentioned * being rendered inadmissable by the age and sex of the patient I happened to recollect a simple and well known fact in acoustics and fancied at the same time that it might be turned to some use on the present occasion The fact I allude to is the augmented impression of sound when conveyed through certain solid bodies as when we hear the scratch of a pin at one end of a piece of wood on applying our ear to the other Immediately on this suggestion I rolled a quire of paper into a sort of cylinder and applied one end of it to the region of the heart and the other to my ear and was not a little surprised and pleased that I could thereby perceive the action of the heart

* Immediate auscultation

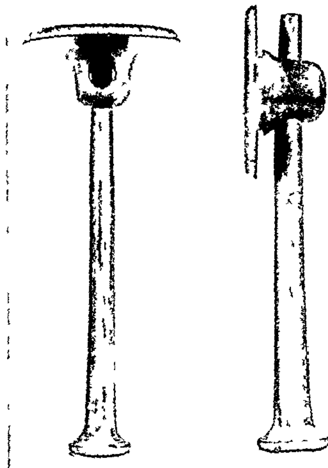


FIGURE 1 Monaural Wooden Stethoscope of the Type Still Commonly Used in European Countries Illustrated are two views showing how the larger ear piece is detached and replaced parallel to the stem for ease of carrying

in a manner much more clear and distinct than I had ever been able to do by the immediate application of the ear From this moment I imagined that the circumstance might furnish means for enabling us to ascertain the character not only of the action of the heart but of every species of sound produced by the motion of all the thoracic viscera

The type of stethoscope Laennec developed from his discovery was the wood monaural which is carefully sketched in his book The monaural stethoscope (*FIG 1*) is still in use in Europe to a considerable degree and has been made of various kind of wood of brass and of hard rubber In this country the binaural stethoscope has been used exclusively for many years The following information regarding binaural stethoscopes was obtained from a handbook entitled *Diagnostic Instruments and Techniques in Medicine* ' The advantage of a flexible stethoscope which would lead the sound to both ears had been recognized for some time in the United States but it was not until 1843 that the first binaural stethoscope was placed on the market No specimen of this instrument is known and its description is very vague In 1855 a stethoscope was introduced by Doctor George P Cammann (1804–1863) of New York City which had a chest piece of ebony *flexible tubing made of spirals of wire covered with layers of silk* dipped in gum elastic and ear tips made of ivory The curved metal ear tubes were fastened to a hinged cross piece and approximation was provided by a strip of elastic web sewn between the tube Although other types were subsequently invented and sold a modification of the original Cammann stethoscope placed on the market about 1890 having a steel spring between the metal ear tubes has remained the standard American stethoscope since that time

CHAPTER II

Importance of Cardiac Auscultation Audio Visual Equipment

NEWER DIAGNOSTIC MODALITIES in cardiology have tended to relegate cardiac auscultation to a position of secondary impor*tance which is unwarranted. Accuracy in the diagnosis of valvular* heart disease is assuming greater importance with the rapid progress of cardiac surgery. For example, when the clinical evaluation of a patient suggests that surgical intervention is indicated, then the auscultatory findings may be of definite aid.

By recognizing the classic to and fro pericardial friction sounds, the diagnosis of pericarditis can be made without the use of the electrocardiogram and at times may be the sole diagnostic sign of pericarditis. This is particularly true when the clinical picture and the electrocardiographic findings are obscured by associated pathologic changes, as in myocardial infarction.

There is no question that the electrocardiogram is the most accurate method of diagnosing cardiac arrhythmias, but its use is not always possible. For example, one of the common cardiac emergencies is the sudden onset of a cardiac arrhythmia. The physician must make a prompt decision regarding therapy and thus identification of the arrhythmia is important. *By utilizing the history,* cardiac auscultation and the effect of carotid sinus pressure this decision can be made with a high degree of precision and proper therapy initiated. It is, however, advisable to check this presumptive diagnosis electrocardiographically. With experience the recognition of various cardiac arrhythmia becomes simpler by means of careful cardiac auscultation.

The evaluation of myocardial function is admittedly a diffi

cult problem but proper interpretation of certain auscultatory findings allows one to arrive at certain conclusions The finding of a diastolic gallop rhythm, for example, usually indicates a failing myocardium A changing relationship between the intensities of the second aortic and second pulmonic sounds may indicate the development of pulmonary hypertension secondary to a failing left ventricle As a case in point in a known arteriosclerotic or hypertensive cardiac the development of an accentuated second pulmonic sound suggests impending left ventricular failure

With careful auscultation differentiation of a physiologic third heart sound the opening snap of the mitral valve in mitral stenosis duplicated sounds and systolic clicks becomes possible Under certain circumstances confirmation of these additional sounds may require additional instrumental studies

METHOD AND EQUIPMENT

Since 1946 we have been *intere ted in the problem of teach* ing auscultation of the heart by the u e of audiovi ual equipment[7] *Over a period of years many refinements have been made and* undoubtedly the future will bring further advances

Our primary concern has been to faithfully reproduce what the physician is accustomed to hear through his stethoscope This *is pos ible by the u e of a carefully designed apparatus* consisting of a microphone applied to the chest of the patient a special amplifier with suitable filters and an electronic stetho cope for each observer (FIG 2) The number of individuals listening simultaneously is limited only by the number of available stethoscopes In this way many physicians may listen to a patient at the same time This is acceptable to the patient since the period of examination is materially reduced and repeated examinations by one individual after another become unnecessary One in tructor is adequate to conduct the entire group

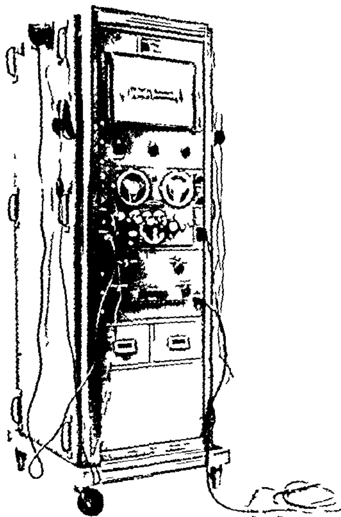


FIGURE 2 Educat onal Card oscope This is a large instrument de signed pr mar ly for teaching purposes It is used to demonstrate the instantaneous stethogram electrocard ogram or combinations of the two while a large g oup l stens to the heart sounds through individual electronic stethoscopes A tape recorder makes a permanent ecord ing of heart sounds In our group th s instrument is affectionately called Cyclops (Cambridge Instrument Company)

Reproduction of the heart sounds through a loudspeaker instead of stethophones is sometimes used but in our experience individual stethophones have been decidedly more satisfactory.

In addition to the accurate reproduction of the sounds we have found their simultaneous visualization (stethogram *) to be of the greatest importance. This is accomplished by feeding the output of the sound amplifier into an oscilloscope with a screen which retains the image of the electron beam for a few seconds producing a persistent pattern of the stethogram. Other phenomena such as the electrocardiogram may also be visualized simultaneously with the sounds.

It has been our experience that the *simultaneous delivery of sound to the ear and of the visual pattern to the eye has great advantage over either alone*. The eye cannot discriminate such phenomena as breath sounds, movement of the microphone on the chest wall, hair and skin noises, although the experienced ear does this readily. On the other hand the visual pattern is much better for analysis of time relationships in the cardiac cycle, and low frequency, low intensity sounds may occasionally be seen which are heard only with difficulty or not at all even after their presence and position in the cardiac cycle have been noted by the eye.

Since patients are not always available for the demonstration of all types of auscultatory phenomena a magnetic tape recorder of special design † has been developed to reproduce heart sound

* The terms phonocardiogram and stethogram are often used interchangeably. It is our opinion that the word phonocardiogram implies the simultaneous recording of the stethogram and the electrocardiogram.

† While the *high quality* tape recorders commercially available can reproduce heart sounds accurately, a suitable microphone is required. In addition no filter system is present in such recorders and their high tape speed is unnecessary in the frequency range of heart sounds. In our experience the type of tape recorder used in homes or offices generally does not have sufficient fidelity below 100 cycles per second to reproduce heart sounds accurately.

accurately Endless loops of tape permit the continuous demon stration of any recording for as long or short a period as neces sary for complete understanding FIGURE 3 illustrates a new

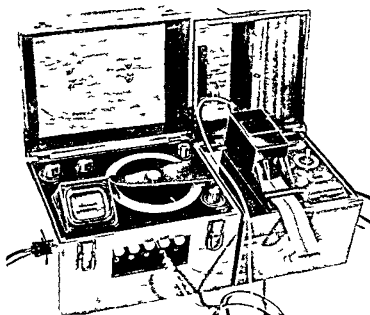


FIGURE 3 Audio Visual Heart Sound Recorder with Direct writer ECG This new device was designed and developed for use by indi viduals or small groups and is portable It enables the physician to study the auscultatory findings in patients by the combined audio visual method Amplification and selective filtration make the percep tion of difficult sounds and murmurs easy In addition permanent sound recordings on discs of magnetic tape can be made and filed with a patient s chart for future reference A direct writer electro cardiograph can be used with this recorder to put on the tape a signal coinciding with the R wave of the electrocardiogram thus identifying the first heart sound (FIG 49) The electrocardiogram or the stethogram or one superimposed on the other can be visualized on the oscilloscope Sound recordings of the stethograms used in th s book may be obtained for reproduction on this instrument from Cambridge Instrument Company New York City

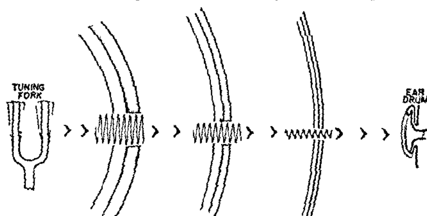
portable instrument designed and developed for the individual physician or small groups interested in accurate auscultation

With tape recordings it is possible to reproduce with fidelity any sound which has been previously recorded Such findings as pericardial rubs and other transitory phenomena are thus always available for teaching Tape recordings are also invaluable in following the progress of a patient over a period of time before and after operative procedures on the heart and for correlation with postmortem findings In addition we feel that this method will be of tremendous value to industrial health departments school physicians and Life Insurance Companies

CHAPTER III

The Physical Principles of Sound

EVERYONE IS FAMILIAR with the fact that when a tuning fork is sharply tapped a pure tone is heard by the ear The pitch of this tone is dependent upon the mass of the tuning fork and the length of the arms As the arms of the tuning fork vibrate the adjacent particles of air are displaced back and forth a certain number of times per second (FIGURE 4) This displacement initiates a wave composed of forward compression of air particles



INTENSITY DIMINISHES AS SQUARE OF DISTANCE

FIGURE 4 *Transmission of Sound Waves from a Tuning Fork The wave spreads in all directions from the point of origin and the tuning fork is thus the center of the wave fronts Since the intensity of sound diminishes according to the square of the distance the waves become less intense as they approach the ear The center inserts demonstrate the diminishing intensity of a pure cycle frequency of 20 cycles when recorded in the same manner as the stethograms in this book The schematic nature of this diagram does not accurately reflect the diminution of intensity with the square of the distance*

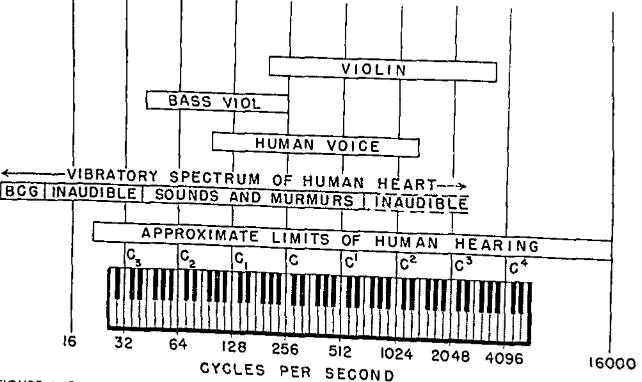


FIGURE 5 Frequency Range of Cardiac Vibrations and Musical Sounds as Related to the Piano Keyboard The diagram indicates the low frequency characteristics of the vibratory spectrum of the human heart Dotted lines represent the unexplored higher frequency vibrations of low intensity which may or may not be of clinical significance BCG represents the ballistocardiogram The BCG and inaudible range at the low frequency end of the spectrum are inaudible because of the characteristics of the human hearing mechanism (FIG 11) but are of high energy at the present time they are of unknown clinical significance The lower end of the vibratory spectrum approximates zero

followed by a similar rarefaction This to and fro motion is in turn transmitted to the surrounding air and a sound wave is propagated in all directions provided there is no physical interference to its spread The phenomenon is similar to the ripples which result from dropping an object into a body of still water

When the sound wave (which decreases in intensity by the square of the distance) reaches the ear the ear drum resonates or vibrates at the same frequency as the tuning fork and this vibration is then perceived by the human hearing mechanism as a pure tone Unless the vibrations are continuous from the source the ear drum is damped in such a manner that it ceases to vibrate immediately otherwise ringing and other peculiar effects resulting from after vibrations of the drum would occur

The quality of a sound is determined by the *pitch* the *intensity* and the number of *harmonics*

The *pitch* is determined by the number of vibrations per second of the sound source This is referred to as cycles per second or cps As an example middle C on the piano has a fundamental tone at the frequency of 256 cps and low C (an octave lower) a frequency of 128 cps The lowest note on the piano scale has a fundamental tone of about 30 cp and the fundamental of the highest key is about 4600 cp FIGURE 5 illustrate the range of pitch of several different varieties of sound

Sounds of the same cycle frequency may be loud or faint and the *intensity* is dependent upon the magnitude of displacement of the air particle by a sound wave The more forceful the displacement the greater will be the magnitude of the resultant vibration of the ear drum and the louder the sound is interpreted by the human hearing mechanism

The following table (FIG 6) adapted from Steven and Davis[8] gives some idea of the comparative intensities of sounds in decibels *(a measure of intensity used by sound engineers)*

From this table it can be appreciated that even in a very quiet room there are several decibels of noise which may interfere decidedly in auscultation particularly since heart sounds are so near the threshold of hearing It is obvious that many murmurs are missed during auscultation under unfavorable circumstance

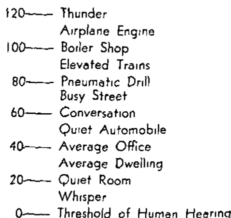


FIGURE 6 Common Sounds Measured in Decibels

Most sounds are not pure tones unless generated by a tuning fork or other special device Actually they are mixtures of the fundamental tone plus *harmonics* which are multiples of the frequency of the fundamental tone As an example if the piano string of middle C vibrates as a unit it will have a frequency of 256 cps It may in addition vibrate in halves and the vibration of each half is twice the fundamental or 512 cps The same may occur for each quarter eighth etc of the length of the string (FIG 7) These harmonics blend with the fundamental tone to give a mixed sound which has a special character Harmonics vary greatly with the manner in which the sound is produced so that middle C on the piano has a different character from the middle C of a violin or the middle C of a wind instrument

The human voice is replete with harmonics which are introduced by the nasal passages and sinuses acting as resonating chambers and their variation from person to person accounts for the individuality of voices

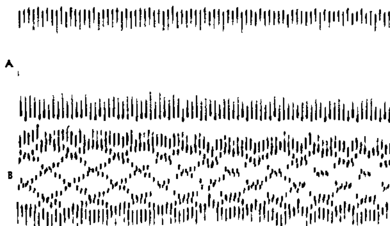


FIGURE 7 (A) Illustrates a pure tone of 64 cycles per second (B) Illustrates the same basic 64 cycle tone with the fourth harmonic (256 cycles) added at one fifth the intensity In listening to this combination the harmonic tone is much the louder even though its intensity is only 20 percent of the basic tone because of the greater sensitivity of the human hearing mechanism at the higher frequency

As far as is known harmonics or overtones are not nearly so important in heart sounds as they are in speech or music the tissues of the body filter out or attenuate the higher frequencies of harmonics (if they are present) in transmission to the surface of the body

CHAPTER IV

Origin of the Heart Sounds

THE ORIGIN of heart sounds has been of unusual interest and controversy since the introduction of mediate auscultation by Laennec in 1819.[4] The problem remains relatively unchanged today. The following discussion will attempt to present generally accepted views in a simplified fashion and with emphasis on clinical application rather than theory and speculation.

It is generally agreed that the first heart sound has several components.[9,1] Of these, however, only two—the closing of the mitral and tricuspid valves and the opening of the aortic and pulmonic valves—would seem to produce vibrations of sufficiently high intensity and pitch to be appreciated by the human hearing mechanism. *Preceding and following these audible sounds are low* frequency, low intensity vibrations below the threshold of human hearing. These include residual vibrations initiated by atrial contraction, pre-isometric vibrations of ventricular origin and those caused by ventricular contraction at the onset of systole as well as vibrations resulting from acceleration of blood flow in the great vessels. These may be recorded with sensitive instruments but at present are without clinical significance (FIG. 8).

It is important to recognize that the audible portions of the heart sounds are not the result of vibrations of the valves themselves but rather of vibrations set up in the adjacent blood columns as a result of the sudden opening or closing of the valves. It is these vibrations which are transmitted through the tissues of the body to the surface. A somewhat analogous situation occurs when a faucet on a free flowing water pipe is abruptly closed, producing audible after vibration because of the sudden pressure changes in

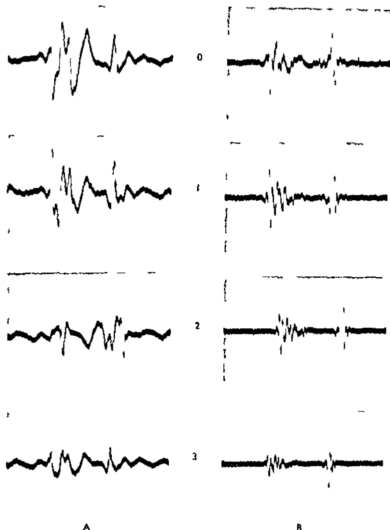


FIGURE 8 Marked Variations Occurring in the Stethogram Depending upon the Type of Instrumentation All recordings were made from the apex of a normal male Those under A were recorded through a microphone which was more sensitive to low frequencies than those under B Zero 1 2 and 3 refer to filter positions which go progressively from low to higher frequency ranges First and second heart sounds respectively appear in each illustration The futility of designating standards for the duration of heart sounds without stating the response characteristics of the recording equipment can easily be appreciated from these comparisons

the water in the pipe There is no evidence at the present time indicating that the muscular contraction of the ventricles contributes to the *audible* portions of the first heart sound

The audible portion of the second heart sound derives from vibrations set up in the columns of blood by sudden closure of the aortic and pulmonic valves There are inaudible components of this sound as with the first sound but they are not clinically significant The exception is the opening of the mitral and tricuspid valves While normally inaudible with the development of stenosis of the mitral or tricuspid valves these vibration become abnormally intense cross the threshold of audibility and are then known as opening snaps

The physiologic third heart sound is rarely heard in adults but in children is noted with fair frequency at the cardiac apex when the heart rate is slow and the patient supine While it is generally accepted that this sound represents vibration resulting from movements of the A V valves and the ventricular walls during the period of rapid ventricular filling in early diastole, recent experiments have suggested that it is caused by the opening of the mitral and tricuspid valves [13]

The fourth heart sound or atrial sound is almost always inaudible This is directly related to atrial contraction but the mechanism is not established In partial or complete heart block it may occasionally become audible

Many determinations of the duration of the first and second heart sounds have been made but these are based on recordings with instruments of widely varying responses Consequently the figures deviate considerably and are of little clinical value (FIG 8) The duration of a heart sound is dependent upon the duration of both the audible and inaudible vibrations but in clinical usage only the period of audible vibrations is measured It can be stated generally that the duration of the first heart sound is about twice

that of the second sound The first sound is lower pitched so that the second sound is heard as the harper of the two FIGURE 9 illustrates the duration timing and audibility of the various heart ounds

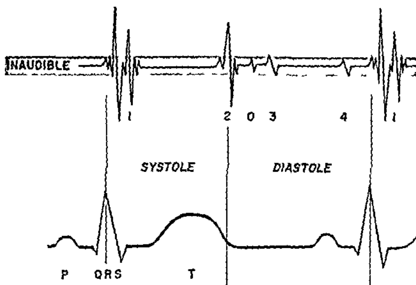


FIGURE 9 Relat onship of the Electrocard ogram to the Variou Heart Sounds with an Indication of the Audible and Inaudible Por tions of These Sounds 1 and 2 represent the first and second heart sounds The gray area repre ent tho e heart sounds which are *usually inaudible but which may be recorded with sensitive low fre quency equipment At times these inaudible sounds become audible* 0 represents the open ng of the mitral and tricuspid valves *In stenosis it often becomes audible and is termed the opening snap* 3 *is the phys ologic third heart sound which may be heard in* hildren and young adults *and* 4 *is the atrial sound which may* be heard *in* heart block or presystolic gallop rhythm

CHAPTER V

Modification of Heart Sounds

Heart sounds are modified in three ways from the points of origin to perception by the human hearing mechanism.[14] The first modification (*A*) occurs in transmission to the surface of the body. The second modification (*B*) is *introduced by the stethoscope and* the third (*C*) by the unusual characteristics of the human ear (FIG. 10).

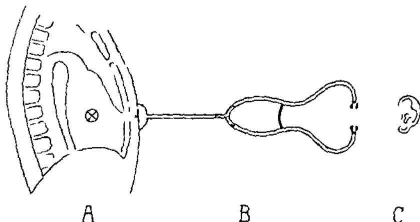


FIGURE 10. Distortion or Attenuation of Heart Sounds. (A) Indicates the area in which changes occur as sound is conducted from the point of origin to the surface of the body, (B) the changes introduced by the stethoscope, and (C) the changes introduced into the interpretation of sound by the human hearing mechanism.

TRANSMISSION THROUGH THE TISSUES

The tissues of the body vary in the conduction of sound. Bone is a good conductor of sound and therefore loud murmurs *can often be heard at a considerable distance from the heart if the*

stethoscope is placed over a bony prominence such as the olecranon of the elbow. Blood and muscle are fair conductors of sound but lung tissue is a poor conductor since it consists of many air spaces which interrupt the transmission of sound. Therefore, even though sounds may be of equal intensity at their points of origin, owing to anatomic variations from person to person they reach the surface of the body at different intensities. Thus, heart sounds are usually loud in people with thin chest walls, while obese people and females with large breasts show marked diminution in the intensity of the sounds. Emphysema and pericardial effusion are among other causes of attenuation. Changes in heart sound will be more fully discussed in Chapter VII.

THE STETHOSCOPE

The type of stethoscope also affects the transmission of sound. There are measurable differences of effectiveness between the bell and diaphragm chest pieces but we have not been able to convince ourselves that one type of stethoscopic chest piece is preferable to another, since a great deal depends upon the individual's hearing and experience. It should be pointed out that the diaphragm type usually have a wider opening and thus the intensity of the sound is greater than with the smaller bell piece.

In addition to the type of chest piece, the length and internal diameter of tubing also modify the transmission of heart sound. Studies by Frederick and Dodge and more recently by Rappaport and Sprague have called attention to the fact that increasing the length of the tubing diminishes the transmission of the higher frequencies. They have also suggested the optimal internal diameter of the system should be 1/8 inch instead of the present 3/16 inch.

It is most important that the earpieces fit the external auditory canal comfortably and that the axis of the earpieces be parallel to

the long axis of the external auditory canal If these precautions are not followed the openings of the earpieces may partially impinge on the wall of the external canal and lower the intensity It is suggested that the curved metal ear tubes be forcibly bent to obtain a proper fit for the individual Richard Cabot aptly stated that It is as rash for anyone to select a stethoscope without first trying the fit of the ear pieces in his ears as it would be to buy a new hat without trying it on What suits A very well is quite impossible for B It is true that one can get used to almost any stethoscope as one can to almost any hat but it is not necessary to do so The ear pieces of the ordinary stethoscope are often too small and rarely too large In case of doubt therefore it is better to err upon the side of getting a stethoscope with too large rather than too small ends" [3]

HUMAN HEARING MECHANISM

The final site of modification of heart sound is the result of the peculiarities of the human hearing mechanism By referring to FIGURE 11 one can see that the human ear does not respond equally at all cycle frequencies As an example at 32 cps the intensity required to reach the threshold of audibility is approximately 100 times the intensity at 256 cps It is unfortunate that the human hearing mechanism is so inadequate for the low frequency heart sound

FIGURE 12 illustrates the energy of heart sound at various frequencies As the cycle frequency of heart sounds decreases the energy level increases markedly to a maximum at about 30 cycles This however is not adequate to compensate for the poor response of the human ear in the low frequency range

It is well known that with increasing age there is a definite loss of hearing acuity However such loss is primarily in the

area above 250 cps which is the range of speech and music while the low frequency response in the range of heart sounds suffers little if at all[8] This phenomenon permits many of the older clinicians to hear heart sounds without difficulty even though their perception of ordinary conversation may be considerably reduced (a fact frequently doubted by interns and residents)

Training is another factor which enters into the appreciation of these low frequency heart sounds The more one learns to recognize sound complexes near the threshold of audibility (i e, difficult to hear) the easier is their recognition on subsequent occasion

The human hearing mechanism can be temporarily deafened by a very loud sound and thus "dead spot occur If a heart sound is followed closely by a loud crescendo murmur usually the ear cannot dissociate the two If the process is reversed however and if the sound follows a decrescendo murmur the ear hears both quite well The point has been easily demonstrated by playing some of our tapes backward (FIG 13) A similar phenomenon occurs when exposure to a brilliant flash of light results in momentary blindness

Environmental noise is a frequent cause of failure to hear near threshold sounds and is a common source of error in auscultatory findings In the average quiet room the noise level is 20 decibels or more (FIG 6) most offices in busy urban areas have a considerably higher noise level making accurate auscultation of the heart difficult if not impossible under these circumstance

MAJOR FREQUENCY RANGES OF HEART SOUNDS AND MURMURS

Williams and Dodge[14] in 1925 reported detailed work on the frequency ranges of heart sounds and murmurs and their work

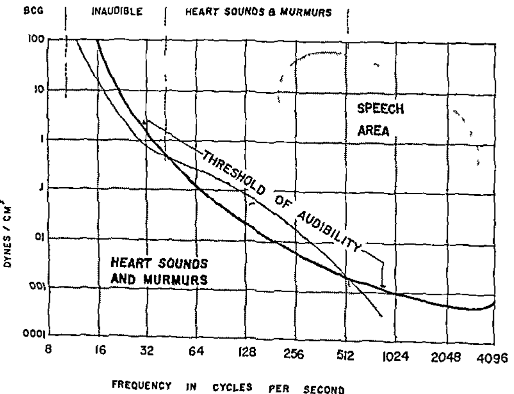


FIGURE 11 This graph indicates an average threshold of audibility and shows how small a portion of the vibratory spectrum of the heart lies above the threshold of audibility Since there may be considerable variation in the threshold of audibility from person to person it is easy to see why differences of opinion are constantly occurring about heart sounds and murmurs

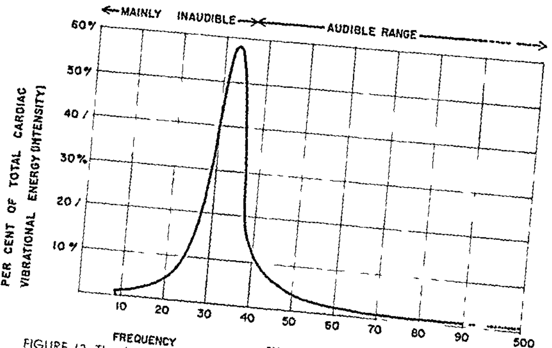


FIGURE 12 The shaded area of the graph shows the estimated energy or intensity of heart sounds at variou frequencies the maximum is about 30 cycles Unfortunately the area below 50 cycles is largely unexplored and is e timated to include 90 percent of the total energy of heart sounds Constructed from data by Williams and Dodge [14] and Folger Smith and Fleming [18]

has never been improved upon We have been interested in the *auditory components* of sounds and murmurs and have investigated them by using a very sharp variable filter While an auditor listened carefully to a specific sound the filter was adjusted without his knowledge to the narrowest band *without noticeable audi*

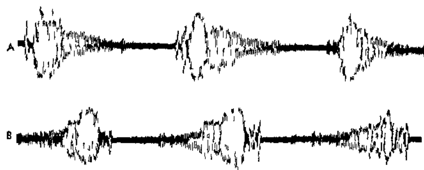


FIGURE 13 Musical Diastolic Murmur of Aortic Insufficiency (recorded at the fourth interspace near the left sternal border) The history and clinical findings were typical of a ruptured cusp of the aortic valve This murmur was confused on auscultation with a systolic murmur and was so designated by the admitting intern An interesting phenomenon was noted when it was visualized on the oscilloscope which showed a distinct second sound that could not be heard because of the loud murmur immediately following (A) When a tape recording was made and the tape then played backwards (B) the second sound followed the murmur of course and the sound could thus be readily heard on auscultation because of the period of silence which followed We have frequently demonstrated this phenomenon which illustrates one of the inadequacies of the human hearing mechanism

tory change to him We found in general that most normal heart sounds fell in the range of 60 to 100 cps, although the third heart sound when heard is somewhat below this range Most systolic murmurs were in the range from 80 to 120 cps while the low pitched diastolic rumbles of mitral stenosis were mainly heard between 40 and 100 cps The slightly higher frequencies of murmurs of aortic and pulmonic insufficiency lie mainly between 100

and 200 cps Note that although these murmurs are generally referred to as high pitched they have in reality a frequency range only slightly above the other cardiac sounds They appear much higher in pitch and louder to the human ear because of the greater sensitivity of the hearing mechanism at this slightly higher frequency band

One must remember that these are generalizations and that the heart sounds and murmurs have ranges well on either side of the figures listed above We are here attempting to show only the ranges which are of greatest significance in clinical auscultation of the heart

THE STETHOGRAM

The stethogram is a visual record of the vibrations produced by the heart The form of the stethogram is determined by the frequency characteristics of the recording equipment and it is regrettable that much of the available data are inaccurate because investigators were not aware of limitations of the equipment used

Three types of stethograms have been classified by Rappaport and Sprague[9] which are arbitrarily defined as linear stethoscopic and logarithmic

The linear stethogram is a low frequency recording of the vibrations from the chest wall which are in the main inaudible and resemble what one feels by palpation The stethoscopic stethogram is a recording of the vibrations as delivered to the ear by the average stethoscope This is in a higher frequency range but still may contain frequencies below the threshold of hearing The logarithmic stethogram is a recording only of frequencies which the human hearing mechanism is capable of interpreting as sound and has the same general response curve as does the human ear

We have approached the problem of stethography in a somewhat different manner. We use equipment which has a known and constant response over the frequency range of cardiac vibrations. By utilizing variable electronic filters, we can record any portion of the vibratory spectrum of the heart (FIGS. 5 and 8). In this way we are not limited to any arbitrary type of stethogram but rather select the range most important to the particular problem at hand. The vibratory spectrum of the heart is a wide one, consisting at one end of very low inaudible frequencies which are similar to the vibrations recorded by the ballistocardiogram and the apex cardiogram. At the other end are the higher frequencies which are in the audible range. Between these two extremes of the spectrum is an inaudible band that is basically unexplored.

The stethogram has certain advantages as well as disadvantages. If it is recorded simultaneously with an electrocardiogram or other adequate timing mechanism, it can be helpful in the timing of sounds and murmurs at fast heart rates. The stethogram also provides a permanent visual record of the sounds. It is dangerous to make diagnoses from the stethogram alone, however, since the eye is not sensitive to pitch and cannot differentiate artefacts and extraneous sounds as does the experienced ear. For this reason it is our practice to combine the sound with its simultaneous visualization on the oscilloscope so that the advantages of both vision and hearing are utilized.

CHAPTER VI

Routine of Auscultation of the Heart

The purpose of cardiac auscultation is to determine the rhythm of the heart the character of the heart sounds and the presence of abnormal sounds and murmurs

The ear tips of the stethoscope should fit well A quiet room is essential The patient should be examined in the upright supine and left lateral positions Effects of exercise and respiration should be determined Exercise usually increases the intensity of heart sounds and murmurs Because of the closer approximation of the heart to the surface of the body they are usually better heard in expiration than in inspiration

The valve areas for auscultation (FIG 14) are (1) the aortic area in the second right intercostal space near the sternal border (2) the pulmonic area similarly situated in the second left intercostal space (3) the tricuspid area at the lower end of the sternum and (4) the mitral area located at the cardiac apex (an area varying therefore with the position of the latter) It should be remembered that the anatomic valve areas are more closely grouped than the auscultatory valve areas (FIG 14) and lie for surface representation roughly within a small area near the junction of the left sternal border at the fourth interspace In addition to the auscultatory valve areas in particular the examination should also include the precordium in general since there is considerable individual variation in the transmission of sounds and murmurs

It has been our experience that the examiner can best orient himself to the timing of sounds by initiating auscultation at the

aortic area and progressing to the pulmonic area then along the left sternal border to the tricuspid area and finally laterally to the cardiac apex It is necessary to listen to several cardiac cycles at

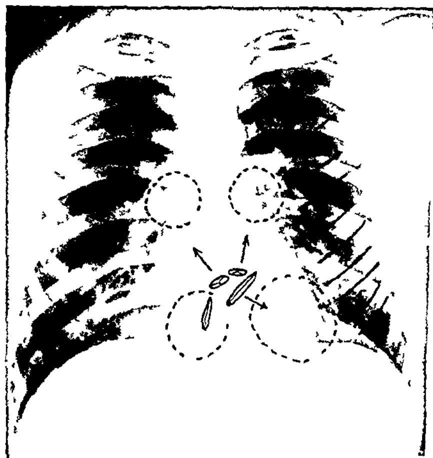


FIGURE 14 Location of Valves and Auscultatory Areas This illustration shows the close relationship of the actual valves as projected in the frontal plane of an x ray film Dotted lines indicate the areas where the sounds from these valves are usually best heard The second third fourth and fifth interspaces are indicated by numerals along the left cardiac border

each move of the stethoscope in order to be certain of the observations made including respiratory variations

The intensity and timing of heart sounds are of fundamental importance Utilization of these factors aids in the diagnosis of valvular defects and cardiac arrhythmias It is not sufficient merely



FIGURE 15 Normal Heart Sounds at the Mitral Area The first sound is louder and longer than the second at this area The rate in this case was about 60 per minute making the systolic interval definitely shorter than the diastolic interval (Note All stethograms in this book were made by playing tape recordings through a special optical galvanometer with photography at a camera speed of 50 mm per second except where specifically stated under individual illustrations Since the illustrations have been variously reduced for publication these figures cannot be used directly for measurements but wherever the time intervals are important they will be included in the captions Equipment provided through the courtesy of the Cambridge Instrument Company)

to listen to the heart sounds in all valvular areas but attention must also be directed to differentiating the first and second sounds and noting changes in their character and intensity at the various valve areas

The identification of the first and second sounds can be made in several ways Acoustically the first sound at the apex is louder longer in duration and lower in pitch (FIG 15) The first sound can be identified by the simultaneous palpable or visual pulsation

of the carotid artery This technic is unsatisfactory when tachycardia is present One can also differentiate the heart sounds by noting their relationship to the longer diastolic interval Since diastole is longer than systole at rates approximately below 100 the sound heard after the longer interval is obviously the first sound

Occasionally the first and second sounds are most easily timed in a certain area By moving the chestpiece of the stethoscope a short distance at a time from this area to other points the sounds may be properly identified over the entire precordium This maneuver has been termed inching by Levine and Harvey [10]

After evaluating the heart sound the examiner should concentrate on the systolic and diastolic intervals to detect additional sounds and murmurs In the course of this routine the rhythm of the heart will have been noted The above description gives the impression of a lengthy examination but fortunately less time is required than the description suggests providing the examiner establishes a routine

CHAPTER VII

Variations of Heart Sounds in Health and Disease

THE FIRST AND SECOND HEART SOUNDS

IN CHAPTER IV mention has been made of the valvular origin of the first and second heart sounds FIGURE 16 summarizes the changes in their intensity Cardiac output and rate of increase in intraventricular pressure are factors that influence the intensity of the first and second heart sounds Obesity emphysema pericardial effusions and other factors tend to diminish the intensity of heart sounds while conditions such as thyrotoxicosis anemia beriberi tachycardia of emotional instability and exercise produce accelerated blood flow and result in accentuated heart sounds

The diminution of the heart sounds in myocardial infarction deserves discussion The term tick tock has been applied to the heart sounds often heard in this condition particularly in the first few days when the heart rate is apt to be rapid and the length of systole and diastole about equal While there may be diminished intensity of both the first and second sounds at the apex there is proportionally a greater reduction in the first sound The mechanism of this change is obscure but is probably related to fast rate and sluggish acceleration of intraventricular pressure

The most important cardiac factor responsible for the loudness of the first sound is the position of the A V valves (mitral and tricuspid) at the time of ventricular systole This is determined by the time interval between atrial and ventricular contraction which is measured by the P R interval of the electrocardiogram It has been demonstrated that usually when the P R interval is short

FIGURE 16 Alterations in the Intensity of Heart Sounds

First and Second Heart Sounds

Accentuated	Diminished
1 Thin chested individuals and children	1 Thick chested individuals
2 Hyperthyroidism	2 Myxedema
3 Fever	3 Myocardial infarction
4 Anemia	4 Shock
5 Exercise	5 Pericardial effusion
6 Emotional tension	6 Constrictive pericarditis
	7 Terminal states
	8 Obesity
	9 Cardiac decompensation (variable)
	10 Emphysema

Changing Intensity of First Sound (Apex)

1 Complete heart block
2 Atrial flutter
3 Ventricular tachycardia

Changing Intensity of First and Second Heart Sounds

1 Atrial fibrillation
2 Extrasystoles

First Sound (Apex)

Accentuated	Diminished
1 Mitral stenosis	1 Prolonged P R interval
2 Short P R interval	

Second Pulmonic Sound

Accentuated	Diminished
1 Pulmonary hypertension (a) Mitral stenosis (b) Left ventricular failure (c) Pulmonary heart disease (d) Congenital heart disease (certain types)	1 Pulmonic stenosis (Valvular type)

Second Aortic Sound

Accentuated	Diminished
1 Hypertension	1 Aortic stenosis (valvular type)
2 Dilatation of ascending aorta (a) Syphilitic aortitis (sometimes tambour sound) (b) Arteriosclerosis (occasional)	

(below 14 second) the A V valves are widely open and low in the ventricular cavity With ventricular contraction these valves rapidly traverse a wide arc of closure which results in an accentuated first sound Conversely when the P R interval is long (about 20 second) the A V valves have floated upwards to an almost closed position and thus traverse a small arc in closing with ventricular contraction resulting in a diminished first heart sound Thus in first stage heart block the first sound is usually reduced in intensity Where the P R interval is short the first sound is usually accentuated although there are exceptions In atrial flutter complete heart block and ventricular tachycardia one frequently hears variations in the intensity of the first heart sound These are dependent on the varying time relationships between atrial and ventricular contractions

There is of course the phenomenon of an impure sound but no general agreement as to what constitutes it At times a sound particularly the first may appear prolonged and somewhat duplicated yet does not have the quality or duration usually ascribable to a murmur To this the term impure sound is applied

In adults the second aortic sound is usually louder than the second pulmonic sound In childhood and adolescence the converse is true It must be remembered however that an accentuated second aortic sound may be transmitted to the pulmonic area and vice versa Increased pressure in either system leads to an accentuation of the second sound over the respective valve area Anatomical changes too most commonly produced by syphilis or arteriosclerosis may result in accentuation of the second sound In syphilis a change in the character of the second aortic sound referred to as a tambour sound may occur it is the result of accentuation plus a musical quality

The finding of an accentuated second pulmonic sound in an adult should make the examiner search for causes of pulmonary

FIGURE 17 Extra Heart Sound

	Point of Maximum Intensity	Time Relation	Character	Remarks
Duplicated 1st sound	Apex	—	Both sounds similar One may be louder than the other	Commonly occurs in normal hearts No significance
Duplicated 2nd sound	Base	—	Both sounds similar One may be louder than the other	Must be differentiated from the opening snap Heard in both normal and abnormal hearts
The opening snap in mitral stenosis	Th rd or fourth interspace at left sternal border	Approximately 08 second after 2nd sound	Sharp snap or clicking sound Loud or faint	Must be differentiated from duplicated 2nd sound and physiologic 3rd sound Common in m tral stenosis
Physiologic 3rd sound	Inside apex usually 4th interspace	Approximately 14 second after 2nd sound	Usually faint low pitched	Must be differentiated from duplicated 2nd sound the opening snap and gallop rhythm
Atrial (4th sound)	Apex or inside apex	0 08–0 14 second after beg nning of P wave	Faint and low pitched	Occasionally heard in heart block
Systolic click	Apex or inside apex	Between 1st and 2nd sound Often varies with respiratory phases	Sharp snap or clicking sound	No pathologic ignificance Mechanism unknown

hypertension This may result from left ventricular failure mitral stenosis pulmonary disease or certain types of congenital heart disease In patients with arteriosclerotic or hypertensive heart disease notation of the relative intensities of the second aortic and pulmonic sounds should *always be made because the advent of* left ventricular failure may be detected by an increasing intensity of the second pulmonic sound At times this increase is so marked that the second pulmonic sound becomes louder than the second aortic sound Aortic area systolic murmurs arising from arteriosclerotic dilatation of the aorta are usually associated with either a normal or accentuated second sound while the systolic murmur of aortic stenosis classically is associated with a diminished or absent second aortic sound

In dextrocardia the heart sounds over the left side of the chest in the usual location of the apex are distant but on the right side of the chest the sounds are normal

ADDITIONAL OR EXTRA HEART SOUNDS

The group of extra heart sounds (FIG 17) include (1) duplicated first or second sounds (also known as split or reduplicated sounds) (2) systolic click (3) opening snap of the mitral or tricuspid valves in stenosis (4) physiologic third heart sound (5) atrial sound (fourth heart sound) (6) third sound of diastolic gallop rhythms and (7) other precordial sound

The physiological third heart sound and the atrial sound are actually not additional or extra heart sounds but are included here because of their importance in the differential diagnosis of diastolic sounds

DUPLICATED SOUNDS Splitting of the first or second sound frequently occurs in normal hearts While pronounced

duplication of the first sound (FIG 18) is uncommon minor degrees are frequently heard in normal hearts at the apex Asynchronous closure of the mitral and tricuspid valves is the usual explanation offered for this condition but adequate experimental proof is not available at the present time An apical duplicated first sound is benign and important only in that it may be confused with the presystolic murmur of mitral stenosis Actually this



FIGURE 18 Duplicated First Sound at the Mitral Area This recording is from a patient who wa referred to us with the mistaken diagnosis of a presystolic murmur There were no other abnormal ascultatory cardiac findings and this duplication has persisted for years The error in diagnosis is underlined by the unfortunate fact that this patient has had an anxiety state re ulting from the mistaken interpretation

error should not be made if one recalls the following diagnostic features

Duplicated sounds tend to be sharp and individual and lack the crescendo buildup of a presystolic murmur Absence of such confirmatory signs of mitral stenosis as an accentuated second pulmonic sound an accentuated first sound at the apex an opening snap of the mitral valve or a diastolic rumbling murmur will aid in the differentiation

Duplication of the first sound has often been attributed to bundle branch block but in our experience thi association is rare

We do not attribute diagnostic significance to a duplicated first heart sound

Duplication of the second sound is most often heard at the pulmonic area Since it occurs in early diastole it must be dif



FIGURE 19 Time Relationships of a Duplicated Second Sound the Opening Snap of the Mitral Valve in Mitral Stenosis and the Physiologic Third Heart Sound (A) Duplicated second sound (pulmonic area) This duplicated sound was more definitely split on auscultation than appears on the stethogram (B) Opening snap of the mitral valve (fourth interspace at the left sternal border) There is an appreciable period of time about 0 08 second in this case between the second sound and the opening snap which follows The stethogram shows the second sound to be partially split but this was difficult to detect by auscultation (C) Physiologic third heart sound (medial to the cardiac apex) The first sound is followed by a short faint systolic murmur The third sound is widely separated about 0 16 second in this case from the second sound and is of low frequency It is more of a thud than the opening snap which is sharper higher pitched and usually louder on auscultation

ferentiated from the opening snap of the mitral valve in mitral stenosis and the physiologic third heart sound (FIG 19) Asynchronism in the closure of the aortic and pulmonic valves is believed to be the mechanism of production Since the second sound

is of shorter duration than the first a definite duplication is more apparent here than in the first sound

In general duplication of the second sound, particularly in the presence of congenital heart disease may indicate asynchronous functioning of the aortic and pulmonic valves

SYSTOLIC CLICK This is an extra sound occurring between the first and second sounds usually having a 'clicking quality *(FIG 20) It is usually best heard in the supine position at the* apex Most frequently it is midway between the first and second sounds but its timing can be modified by respiration particularly



FIGURE 20 Systolic Click An extra sound with a clicking quality is heard between the first and second sounds and is usually loudest at the mitral area It has no clinical significance and the mechanism is unknown Its position between the first and second sounds may frequently be varied by Muller and Valsalva maneuvers

during the Valsalva of Muller procedures This sound in our experience has no clinical significance its mechanism of production is unknown Confusion with gallop rhythm may occur particularly at a fast rate

OPENING SNAP OF THE MITRAL OR TRICUSPID VALVE IN STENOSIS This is a sharp or snapping sound that occurs shortly after the second sound (FIG 19) Its timing is intermediate between a duplicated second sound and a physiologic third sound In mitral stenosis the opening snap of the mitral valve is best heard in the third or fourth interspace along the left sternal border but frequently is audible over the entire precordium A rumbling diastolic murmur typical of mitral stenosis may follow this sound The opening snap of the mitral valve is of great diagnostic value since it is an obvious auscultatory finding and alerts the examiner to the possibility of mitral stenosis

An opening snap of the tricuspid valve may occur in the presence of stenosis of the tricuspid valve [3] It is loudest in the tricuspid area

THE PHYSIOLOGIC THIRD HEART SOUND This is a faint low pitched sound that also occurs shortly after the second sound but later than the opening snap (FIG 19) It is best heard at the apex with the patient in the supine position when the heart rate is slow It may vary with respiration usually becoming fainter with deep inspiration This is a normal sound heard commonly in children and frequently in young adults It must be differentiated from a duplicated second sound an opening snap and the third sound of diastolic gallop rhythm The absence of gallop cadence tachycardia and signs of heart disease differentiate it from the third sound of diastolic gallop rhythm We are unconvinced that the mechanism of the physiologic third heart sound is understood at the present time Recently Eddelman et al [13] have advanced the concept that this sound is in reality a physiological opening snap of the auriculoventricular valves

THIRD SOUND OF GALLOP RHYTHMS. We restrict the term gallop rhythm to a three sound sequence resulting in a galloping horse cadence in which the extra sound occurs during *diastole*. Generally it occurs only at heart rates above 100 per minute. Gallop rhythms usually indicate a failing myocardium. Potain [1] * called attention to this type of rhythm in 1875, but unfortunately the present rarity of horses has left a generation of physicians unacquainted with gallop (FIG. 21).



FIGURE 21. Gallop of Horses. This is a recording of a horse's gallop to illustrate the three sound cadence. The actual impression of a gallop comes from the regular recurrence of three sounds, each at a different intensity.

Extra sounds occurring during systole may resemble a gallop rhythm. Although aortic and apical systolic gallop rhythms have been described, we have not encountered them and are

* "But what I propose to study at this time presents special characteristics which make it absolutely distinct and which I will emphasize later on. You will understand then, gentlemen, why I wish to reserve exclusively for it the very expressive name created by my revered master Professor Bouillard. This name is marvelously adapted to the sound which it designates and will be particularly useful in distinguishing a group of very singular findings and quite worthy, as you will see, of a special designation." (Our translation.)

inclined to think that the extra sound described in such rhythms is either a short systolic murmur or a systolic click

The technic of inching (page 42) can be helpful in determining the relationship between the extra sound and the first and second sounds In this process the extra sound becomes fainter and at times may disappear thus enabling the examiner to note its relationship to either systole or diastole

There are two main varieties of gallop rhythm The first is presystolic or atrial gallop (FIG 22) in which the extra sound



FIGURE 22 Presystolic or Atrial Gallop Rhythm (mitral area) Compare with FIGURE 21 This has been carefully timed for identification The presystolic sound which accounts for the gallop cadence is louder than either the first or second sounds It is often very difficult by auscultation alone to properly time and identify the extra sound whether it be presystolic or elsewhere in diastole

occurs shortly before the first sound and is evidently the result of atrial contraction Phonocardiographically the extra sound occurs shortly after the peak of the P wave This variety is not heard during atrial fibrillation The second or protodiastolic gallop occurs during the phase of rapid ventricular filling in early diastole and the extra sound is heard shortly after the second sound

Gallop rhythms are best heard at the apex with the patient supine or in the left lateral position Differentiation between the

physiologic third heart sound and the third sound of protodiastolic gallop can be difficult, but the latter occurs in the presence of a fast rate and heart disease, the mechanism of production may be the same [13]

When the heart rate exceeds 100 a shortening of diastole occurs so that the extra presystolic and protodiastolic sounds merge resulting in a "summation gallop This fine differentiation is not of great importance since all gallop sounds have the same clinical significance A rapid rate accentuates the gallop cadence and gallop rhythms are seldom heard at a heart rate below 100

OTHER PRECORDIAL SOUNDS

PERICARDIAL SOUNDS Acute pericarditis and pneumopericardium may produce unusual sounds In acute pericarditis the diagnosis can be made from the classic friction rub which is best described as a rough grating sound heard in both systole and diastole either as a continuous sound or limited to a short portion of each Friction rubs tend to be variable and transitory which helps differentiate them from murmurs They are best heard along the left sternal border but may be audible over the entire precordium In our experience pericardial rubs are frequently unrecognized because they vary widely in intensity pitch duration and quality while the murmur typical of a specific valvular lesion such as the *decrescendo blowing diastolic* murmur of aortic insufficiency, varies little from case to case

In pneumopericardium a 'water wheel murmur' may be heard *This resembles the sounds made by an old fashioned water* wheel and one can hear in systole a peculiar musical clicking associated with splashing sounds It is rare and its intensity is variable Sounds of this type as well as friction rubs are often heard for a few days following cardiac surgery

AIR SOUNDS The presence of air in the mediastinal or pleural spaces has been associated with unusual auscultatory findings over the precordial area In mediastinal emphysema a crunching or snapping sound usually heard in systole can be detected This is known as Hamman s sign Rarely the sound may continue into diastole Left pneumothorax in particular is associated with a variety of sounds that may be heard over the precordium They are usually systolic in time and have been described as clicking crunching crackling and tapping At times these sounds may be audible without the use of a stethoscope

An extremely rare sound that may be heard over the lower precordial area is a splashing sound produced by the cardiac thrust on the diaphragm and stomach when the latter is filled with air and fluid

XIPHOSTERNAL CRUNCH This crunching sound is heard over the xiphoid process of the sternum Despite reports that indicate a high incidence we have never recognized it and believe it must be extremely rare or artefactual in origin

CHAPTER VIII

Definition, Classification and Timing of Murmurs

IN THE EARLY YEARS of the nineteenth century prior to the discovery of mediate auscultation by Laennec in 1816[4] physicians had become interested in immediate auscultation as a means of obtaining information in heart disease. One of the first observations of a murmur was made by Allan Burns of Scotland who described a hissing sound in a case subsequently found at necropsy to have an indurated mitral valve. Burns noted that Rutherford in his Clinical Observation had described a similar case. Since that time our understanding of murmurs has progressed but controversy continues concerning the interpretation of certain of these adventitious sounds. The importance of proper evaluation of murmurs is obvious when one realizes that frequently they are the only abnormal physical finding related to the heart.

A murmur (L *susurrus* whispering) is the auditory perception of vibrations produced by turbulence in blood flow. These vibrations originate in either the heart or great vessels as a result of hemodynamic changes in the cardiovascular system. Classification of murmurs has not been generally satisfactory. We restrict the term pathologic murmurs to those murmurs deriving from cardiovascular disease.

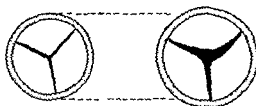
PATHOLOGIC MURMURS

Pathologic murmurs result from the following causes

(a) Deformities of the valve cusps either acquired or congenital

(b) Valvular incompetence In this condition the valve cusps are normal but their closure is faulty resulting in incompetence In the semilunar valves faulty closure results from dilatation of

AORTIC INCOMPETENCE



VIEWED FROM ABOVE

MITRAL INCOMPETENCE

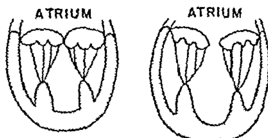


FIGURE 23 Probable Mechanisms in the Production of Aortic and Mitral Incompetence Note that with the enlargement of the aorta the valve ring is stretched and the valve cusps cannot approximate each other during closure resulting in incompetency Mitral incompetence is produced by the stretching of the mitral ring and retraction of the chordae tendineae secondary to enlargement of the left ventricle In both examples the valve cusps are normal in all respects

the ascending aorta or pulmonary artery which stretches the valve ring (FIG 23) In the atrioventricular valves, incompetence results from enlargement of the corresponding ventricle, which retracts the chordae tendineae and papillary muscles and dilates the valve ring (FIG 23)

(c) Nonvalvular congenital cardiac defects

(d) Abnormalities of the great vessels either acquired or congenital

The terms organic (for a, c and d) and relative (for b) are commonly used in classifying pathologic murmurs but we consider them unsatisfactory

NONPATHOLOGIC MURMURS

These murmurs are produced by increased velocity of blood flow cardiorespiratory factors and other unknown mechanisms The terms innocent and functional are frequently used in place of nonpathologic but we consider them unsatisfactory

DESCRIPTION OF MURMURS

Timing	*Duration*	*Intensity*	*Pitch*	*Quality**
Systolic	Short	Very faint	Low	Blowing
Early systolic	Intermediate	Faint	Medium	Harsh
Mid systolic	Long	Moderate	High	Musical
Late systolic	Holosystolic	Loud		Rumbling
Diastolic	Holodiastolic	Very loud		
Early diastolic		Variable		
Mid-diastolic		Crescendo		
Presystolic (late)		Decrescendo		

* There are interrelations between intensity and pitch which determine quality and are at times difficult to separate

EVALUATION OF MURMURS

In order to evaluate murmurs properly attention should be directed to the following features (1) timing (2) duration (3) intensity (4) pitch (5) quality (See chart page 58) Constancy location etiologic history cardiac size and contour and electro cardiographic findings must also be taken into consideration

TIMING All murmurs must be placed in either the systolic or diastolic phases of the cardiac cycle although occassionally they may be continuous (*FIG* 9) This is accomplished by noting the relation of the murmur to either the first or second heart sounds It is customary to subdivide systolic murmurs into early mid or late depending on the phase of systole in which they are heard Diastolic murmurs are subdivided into early mid or pre systolic (late) Diastolic murmurs are pathologic with hardly an exception Systolic murmurs may be pathologic or nonpathologic

DURATION The duration of a murmur is of importance particularly in those occurring during systole Murmurs may be either short intermediate or long If long the term holosystolic or holodiastolic is applied

INTENSITY Attempts have been made to quantitate the loudness of murmurs The range varies from very faint to very loud Levine[19] has quantitated the loudness of systolic murmurs by grading them into 6 groups Grade I applies to the faintest audible murmur while a Grade VI murmur is one that can be heard with the stethoscope just removed from contact with the chest wall This numerical method may be satisfactory to the individual observer but we feel that it is not generally applicable because of individual variations in hearing and the lack of precise standardization

The intensity of a murmur also determines the presence or absence of a thrill. A thrill is actually the palpable manifestation of a murmur, and as the intensity of the latter increases, the greater the probability of a thrill.

Transmission of murmurs was formerly attributed entirely to the direction of blood flow. This was inferred from the murmur of aortic stenosis, which is transmitted upwards, and the diastolic murmur of aortic insufficiency, which is transmitted downwards. It is now recognized that the intensity and point of origin of a murmur are often more important in transmission than the vascular factor, and that bony structures, being good conductors of sound, play an important part in propagation.

The intensity of a murmur may vary during a single cycle as well as from cycle to cycle. When it increases, it is perceived as a crescendo murmur; when it decreases, it is perceived as a decrescendo murmur. At times it may be crescendo-decrescendo, and the term "diamond shaped"[19] has been applied to this type of murmur.

PITCH AND QUALITY. The quality of a murmur is an auditory characteristic depending mainly on intensity and pitch.* In the main, the quality of a sound depends on the relative prominence of the fundamental tone and any harmonics present. However, the harmonics produced by the heart are so low in energy by the time they reach the ear that auditory perception is poor. The quality of murmurs generally depends on gradations of pitch with varying intensity. Thus, a murmur may be blowing, harsh, musical or rumbling. If the pitch is essentially a constant frequency, a musical quality is imparted to the murmur. The

* In general, as applied to heart sounds and murmurs, we consider low pitch to range from 30 to 80 cycles per second, medium pitch from 80 to 120 cycles, and high pitch above 120 cycles.

quality of a murmur is of importance because it often indicates a specific valvular lesion *For example a decrescendo blowing relatively high pitched murmur* diastolic in time indicates insufficiency or incompetence of either the pulmonic or aortic valves *Actually with perseverance one can recognize specific valvular* lesions by the quality and timing of the murmur alone even though it may not be heard in the classic valve area

CONSTANCY The constancy of murmurs is important particularly in the evaluation of systolic murmurs Most pathologic *murmurs are constant Respiration affects pathologic murmurs to* a very limited degree but tachycardia and or changes in position may make certain pathologic murmurs more audible Systolic *murmurs pathologic or nonpathologic are usually accentuated by* exercise

*LOCATION In general murmurs indicating classic patho*logic lesions have classic locations These will be dealt with in Chapter X

The location of a systolic murmur may be of significance in children where the incidence of systolic murmurs in the pulmonic *area is high Most murmurs in this area are nonpathologic Over the aortic area however particularly in adults most murmurs* are pathologic

ETIOLOGIC HISTORY In *evaluating a murmur a history of an etiological* factor for heart disease is important This is particularly true of an apical systolic murmur when there is a history *of rheumatic fever or equivalent* Such *a murmur is suspect* of pathologic origin Continued observation of such a patient may in time develop a *diastolic murmur or changes in the cardiac* silhouette *thus confirming* the pathologic origin of the systolic murmur If repeated examinations over a period of years do not

reveal these changes then one is justified in calling such a systolic murmur nonpathologic About 50 per cent of people with rheumatic heart disease give no history of acute rheumatic fever, and 30 per cent of patients with clinically proved rheumatic fever will show no evidence of rheumatic heart disease ten years after their infection

EVALUATION OF SYSTOLIC MURMURS

The problem of differentiating pathologic systolic murmurs from those of nonpathologic origin is a common one The examiner should have a complete analysis of the murmur

In general the louder murmurs are more apt to be pathologic Pathologic murmurs *are usually constant and have a moderate to* long duration Changes in cardiac contour and size and an abnormal electrocardiogram favor a pathologic origin of the murmur A history of rheumatic fever or its equivalents or other etiologic factors for heart disease also favor a pathologic origin One must always exclude the presence of anemia and hyperthyroidism before attempting evaluation of a systolic murmur An aortic localization of a murmur tends to classify it as pathologic

Actually it is often impossible to evaluate a systolic murmur after a single examination and continued observation utilizing the above factors is essential before arriving at a final decision

CHAPTER IX

Disorders of Heart Rhythm

CORRECT BEDSIDE INTERPRETATION of certain abnormal heart rhythms can be made but it requires careful auscultation and experience For practical purposes the separation of abnormal heart rhythms into tachycardias, bradycardias and irregularities simplifies classification and can be of clinical value When confronted with disorders of rhythm the physician should note the mode of onset, apical and radial rates the regularity the intensity and variations of heart sounds and effects of carotid sinus pressure and exercise It is obvious that not all disorders of the heart beat can be diagnosed at the bedside and our discussion will be limited to those that may most often be determined clinically

TACHYCARDIAS WITH REGULAR RHYTHM

A SINUS TACHYCARDIA This consists of a regular rapid heart action at a rate between 100 and 160 Its onset and retrogression is usually gradual There is generally little or no response to carotid sinus pressure and since there is a normal relationship between atrial and ventricular contractions, the heart sounds have the same relative intensity Recognition of the first and second sounds may be difficult The differentiation may be possible by correlation with the carotid artery pulsation but at rates above 100 it becomes difficult Some examiners have well developed tactile auditory reflexes and can utilize this correlation better than others

B PAROXYSMAL TACHYCARDIAS These tachycardias consist of rapid rates varying between 120 and 250 with an average rate of 160 They are sudden in onset and may stop abruptly

(1) *Paroxysmal Atrial and Nodal Tachycardia* This tachycardia occurs most often in young adults with normal hearts Its onset is sudden and the rhythm is regular at a rapid and constant rate of about 160 An important auscultatory feature is the constancy of the apical rate When carotid sinus or ocular pressure is effective this type of tachycardia is immediately terminated No other tachycardia responds so dramatically to carotid sinus pressure but unfortunately many bouts of paroxysmal atrial and nodal tachycardia do not show this response

(2) *Paroxysmal Ventricular Tachycardia* This type of tachycardia occurs most often in diseased hearts At times it may be responsible for syncopal episodes The ventricular rate may vary between 150 and 250 per minute and is essentially regular as far as can be determined by auscultation Carotid sinus pressure is ineffectual A diagnostic feature occasionally present is the varying intensity of the first heart sound which is produced by the changing relationship between atrial and ventricular contractions

C ATRIAL FLUTTER WITH REGULAR VENTRICULAR RESPONSE In general the atrial rate is 300 per minute with a halved ventricular response Thus when confronted with an apical rate of 150 the examiner should rule out atrial flutter The onset is sudden and duration varies from a few hours to years It is generally associated with heart disease

On auscultation if the ventricular response is constant (2 1 3 1 4 1, etc) no unusual findings can be detected If the block is variable then variations in intensity of the first sound may be

detected When confronted with a regular rhythm at a rate of approximately 150 per minute the effect of carotid sinus pressure is significant if the tachycardia is atrial or nodal in origin a *prompt return to a normal rate may occur or the rate is unchanged* if atrial flutter is present the ventricular rate slows temporarily but quickly returns to the previous tachycardia (during this return varying intensities of the first heart sound may be heard) if ventricular tachycardia is present no response occurs Sinus tachycardia can be slowed by carotid sinus pressure but the effect is temporary

BRADYCARDIAS WITH REGULAR RHYTHM

A SINUS BRADYCARDIA A heart rate below 60 is considered bradycardia This type of slow rate may occur in normal hearts The heart sounds are normal and regular

b HEART BLOCK Complete heart block usually has an apical rate between 30 and 40 per minute and there is complete *dissociation between atrial and ventricular contractions* It occurs most often beyond 50 years of age and is the result of heart disease and often associated with the Adams Stokes syndrome On auscultation one may hear a varying intensity of the first heart sound since there is a variable atrioventricular relationship (FIG 24) At times this intensity change is so loud and sudden it is called bruit de canon and may serve to differentiate complete heart block and sinus bradycardia An additional observation is the appearance of small jugular vein pulsations associated with atrial contraction which will be seen during the long pauses of complete heart block Carotid sinus pressure is without effect on complete heart block *Occasionally in complete heart block* the atrial sounds become audible Amplification with simultaneous

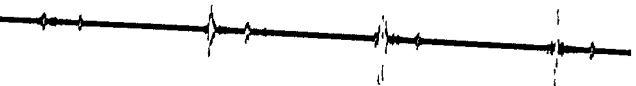


FIGURE 24 Augmentation of the First Heart Sound in Complete Heart Block (25 mm per second) The augmented first sound at the mitral area is due to the changing relationship of the atrial and ventricular contractions and has been called bruit de canon It occurs when an atrial contraction closely precedes ventricular contraction Atrial sounds can often be recorded under these circumstances but none are seen in this record

visualization of the heart sounds makes this observation easier It is often stated that exercise makes atrial sounds more audible but this has not been our experience unless amplification is used

Second degree heart block is difficult to detect by auscultation unless the ventricular respon e is regular and the sound of the blocked atrial beat is audible (FIC 25)

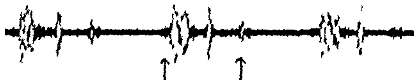


FIGURE 25 *Atrial Sound (25 mm per second) This is referred to as the fourth heart sound and normally it is not heard In this case it was plainly heard and is seen (arrow) in the stethogram immediately before the fir t heart sound be ause of a 2 1 heart block it was also noted as an isolated sound with subsequent ventricular beat missing*

IRREGULAR RHYTHMS

A SINUS ARRHYTHMIA This is an arrhythmia generally phasic where alternately inspiration increases the rate while expiration decrea es it The heart sounds are of normal inten ity Exercise can eliminate this arrhythmia

B EXTRASYSTOLES These may be single or multiple They may originate in either the atria or ventricle They can occur in normal or diseased heart The diagnosis of extrasystoles depend on the udden interruption of a regular rhythm by a beat followed by a pause Generally in an extra sy tole of atrial origin the cycle preceding and the cycle following have a combined

length which is less than two normal cycles making the pause noncompensatory A ventricular extrasystole usually has a compensatory pause, which means that the cycles preceding and following are equal to two normal cycles A nodal extrasystole rarely has a compensatory pause At times the extra beat occurs regularly after a normal beat thus giving a bigeminal quality to the rhythm As a rule in extrasystoles the intensity of the heart sounds produced may vary from reduction to accentuation depending on a series of factors not all of which are known

C ATRIAL FIBRILLATION This tachycardia is paroxysmal in approximately 25 per cent of cases If heart disease is present the fibrillation will eventually become permanent

On auscultation, the rhythm is totally irregular and if apical and radial rates are compared a pulse deficit will be noted In general the ventricular rate in uncontrolled atrial fibrillation is between 130 and 160 per minute The heart sounds continually vary in intensity Multiple extrasystoles at times are difficult to differentiate from atrial fibrillation Exercise usually diminishes the number of extrasy toles but increase the irregularity of atrial fibrillation

D ATRIAL FLUTTER WITH IRREGULAR RESPONSE (See *Atrial Flutter* page 64)

CHAPTER X

Clinical Entities and Their Auscultatory Findings

ETIOLOGIC FACTORS are listed followed by a discussion of the findings. The murmurs described are pathologic murmurs which means that they are caused by abnormalities of the heart or great vessels as distinguished from nonpathologic murmurs which *are not so caused. The lesions are described separately but* actually combinations are the rule

MITRAL STENOSIS

ETIOLOGIC FACTORS: The chief cause is valvular deformity resulting from preceding rheumatic fever. *Congenital mal*formation owing to anomalous development or fetal endocarditis accounts for the remainder of the cases (which are rare)

AUSCULTATORY FINDINGS: The findings vary with the degree of stenosis which explains the variation from case to case. Auscultation usually provides the only evidence of mitral stenosis in its early phase

Among the early signs of mitral stenosis are accentuation of the first sound at the apex and an opening snap of the mitral valve *(FIG 26). The former is practically constant and the latter* is frequently heard. In general they develop at about the same time. The cause of the loud snapping first sound is controversial one suggested cause being a direct increase in intensity due to structural changes in the cusp. Another factor is said to be closure of the valve at a time when the cusp lie deeper in the ventricle

than normally as a result of shortening of the chordae tendineae and papillary muscles and because the increase in intra-atrial pressure may occur later and last longer than in normal hearts. The sound produced by the opening of the mitral valve is normally inaudible, but in mitral stenosis without insufficiency it is frequently heard, in which case it is known as the opening snap of

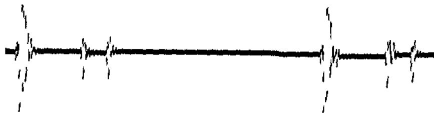


FIGURE 26 Accentuated First Sound and Opening Snap in Mitral Stenosis (medial to cardiac apex) Accepted standards for judging accentuation of sounds are nonexistent but at this level of recording accentuation was definite An opening snap of the mitral valve follows the normal second sound by about 0 12 second in this case Sinus bradycardia was present and the opening snap might have been mistaken for a physiologic third heart sound except for the signs of mitral stenosis—an accentuated pulmonic second sound and at the apex a presystolic and a loud rumbling diastolic murmur These murmurs do not appear in this stethogram which was recorded medial to the apex

the mitral valve. It has been suggested that both the accentuated first sound and the opening snap of the mitral valve are dependent upon the existence of sufficient flexibility in the position of the mitral valve ring so that it moves with the pressure changes in the left atrium and left ventricle much as a sail snaps from one side to the other with sudden changes in the wind. During systole this movement augments the first sound and during diastole accounts for the opening snap. Thus in the selection of cases for

mitral commissurotomy, the presence of an accentuated fir t mitral sound and an opening snap of the mitral valve imply flexibility of the mitral valve

The opening snap is usually best heard along the left sternal border from the second to the fourth interspace There is an appreciable period of time between the second sound and the opening snap which distinguishes the latter from a duplicated second sound At times it may be necessary to differentiate the

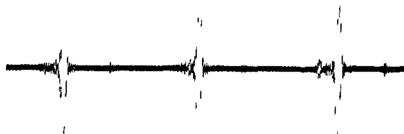


FIGURE 27 Presystolic Murmur of Mitral Stenosis (mitral area) A presystolic murmur is followed by an accentuated first sound The second sound is of very low intensity by comparison and can scarcely be seen This patient was subjected to commissurotomy and had a small stenotic opening which was enlarged by finger fracture S nce *operation the patient has been much improved (Courtesy of Dr* John LaDue)

opening snap of mitral stenosis from a physiologic third heart sound which however appears at a later time and has a quality of a dull thud rather than a sharp snap (FIG 19)

As the stenosis of the mitral valve progresses an apical murmur develops in diastole The duration of thi proce s in general is a matter of years but on occasion can occur in only a few months When the murmur appears in late dia tole it is known a a pres tolic murmur (FIG 27) If the dia tolic murmur occur

earlier in diastole it will have a rumbling character and may be heard in early or mid diastole In our experience a mid diastolic murmur (FIG 28) is not so frequent an early sign of mitral stenosis as is a presystolic murmur When the murmur occurs early



FIGURE 28 Mid diastolic Murmur of Mitral Stenosis (mitral area 25 mm per second) The first sound is of low intensity and is followed by a loud systolic murmur the result of mitral insufficiency The second sound is normal and is followed by a faint mid diastolic murmur (arrow) The absence of an accentuated first sound is probably the result of the mitral insufficiency which is more pronounced in this case than the mitral stenosis This record was made in 1948 at which time the mid diastolic murmur was barely audible with the stethoscope but with selective filtration and amplification it was easily heard At the present time (1955) the patient has a loud holodiastolic murmur with presystolic accentuation as well as the systolic murmur His symptoms have progressed accordingly but because of the mitral insufficiency commissurotomy is deemed unwise

in diastole, it begins very shortly after the second sound (FIG 29) Various combinations of these murmurs exist so that some cases will have a murmur taking up the entire diastolic period (holodiastolic) (FIG 30) It is well to remember that with regular sinus rhythm a presystolic murmur need not be invariably present in mitral stenosis The murmur of this lesion may be localized to a small area just medial to the cardiac apex and may *be readily missed if this is not kept in mind* In the earlier phase

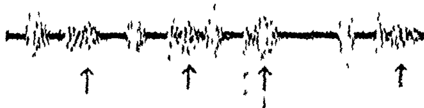


FIGURE 29 Early Diastolic Murmur of Mitral Stenosis with Atrial Fibrillation (mitral area 25 mm per second) *The first sound is accentuated and followed by a normal second sound An opening snap follows the second sound by about 0.07 second and immediately following the snap there is a low pitched early diastolic murmur* (arrow) *which on auscultation had a rumbling quality The opening snap in this case cannot be differentiated by ear from the murmur but it is seen on the stethogram*

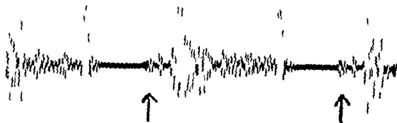


FIGURE 30 Holodiastolic Murmur of Mitral Stenosis (mitral area) *There is an accentuated first sound followed by a silent interval which represents systole The second sound* (arrow) *at the end of this interval is faint by comparison and is followed by an opening snap after which a very loud holodiastolic murmur is present There is accentuation in early diastole (protodiastole) This was heard as a very loud rumbling murmur The second sound and opening snap are of such low intensity that they were not differentiated on auscultation Mitral commissurotomy was performed on this patient with marked benefit For the postoperative recording see* FIGURE 34



FIGURE 31 Presystolic Murmur Separated from the First Sound in Mitral Stenosis (mitral area) The presystolic murmur is definitely separated from the accentuated first sound because of a lengthened period between auricular and ventricular contraction The P R interval was 24 second The loud first sound is followed by a murmur during systole which in this case was transmitted from the aortic area as the result of aortic stenosis (see FIGURE 40 for aortic stethogram in this case) An opening snap is present about 0 10 second after the second sound



FIGURE 32 Disappearance of Presystolic Murmur with Atrial Fibrillation (mitral area) (A) Presystolic murmur followed by an accentuated first sound An opening snap follows the second sound and is in turn followed by a short early diastolic murmur (B) Same patient a few days later after the onset of atrial fibrillation The presystolic murmur has disappeared (A presystolic murmur results from atrial contraction just preceding ventricular contraction and this is abolished by atrial fibrillation)

of development this murmur may not be heard unless the patient is examined in the left lateral position or after exercise or both but later it may be heard in any position At times a series of rapid coughs can accomplish the same effect as exercise

The presystolic murmur is caused by increased blood velocity through a stenotic opening as a result of atrial contraction The crescendo quality of the murmur is due to its proximity to the first sound It loses this quality if the interval between atrial and ventricular contraction (P R interval) is sufficiently long to separate the murmur from the first sound (FIG 31) The presystolic murmur disappears with the onset of atrial fibrillation since there is no coordinated atrial contraction Reappearance can be demonstrated in cases of spontaneous conversion to normal sinus rhythm or when conversion results from the use of quinidine or other drugs (FIG 32) The accentuated first sound and the opening snap of the mitral valve are not altered by the onset of atrial fibrillation although with fast ventricular rates the snap may be difficult to hear A certain confusion is encountered in patients with rapid atrial fibrillation in whom a presystolic murmur is believed to be heard In our opinion this is not a true presystolic murmur but a mid diastolic murmur appearing in close approximation to the first sound when the irregularity results in a shortened diastolic period (FIG 33)

An advanced degree of mitral stenosis results in pulmonary arterial hypertension which in itself produces accentuation of the pulmonic second sound sometimes with duplication and occasionally with a Graham Steell murmur (FIG 46) The latter is a murmur of pulmonic incompetence due to dilatation of the pulmonic valve ring At times differentiation between an early aortic insufficiency and the Graham Steell murmur is very difficult Cardiac contour and peripheral manifestations of aortic insufficiency help in making this differentiation possible

Extreme cases of mitral stenosis may not show the classic findings particularly in the presence of congestive failure, atrial fibrillation, or a large left atrial thrombus. Autopsied cases have been recorded which had not demonstrated an accentuated first sound or a murmur.

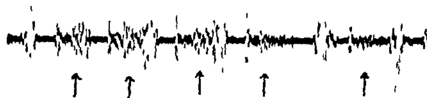


FIGURE 33. *Pseudo presystolic Murmur of Mitral Stenosis in Atrial Fibrillation (mitral area 25 mm. per second). This is a mid diastolic murmur (arrow) quite variable in intensity which comes into close approximation to the first sound whenever the irregularity of atrial fibrillation results in a short diastolic interval. In this stethogram the accentuated first sound and the second sound can be easily identified because of the silent systolic interval between them. The effect of varying diastolic interval on the mid diastolic murmur is apparent.*

SELECTION OF CASES FOR MITRAL COMMISSUROTOMY. There is still some controversy as to whether or not mitral surgery is indicated in asymptomatic cases. In evaluating patients for mitral commissurotomy careful auscultation is an important modality. When specific clinical symptoms exist the finding of an accentuated first mitral sound, an opening snap of the mitral valve, accentuation of the second pulmonic sound and a diastolic murmur typical of mitral stenosis make the case ideal for surgery, particularly if the cardiac contour and electrocardiogram are compatible with the diagnosis of mitral stenosis. Occasionally

certain cases may in addition have an apical or basal systol murmur In our opinion if this does not indicate a lesion pr ducing hemodynamic changes as evidenced by left ventricul enlargement on fluoroscopy or in the electrocardiogram surger may be recommended A mild degree of aortic insufficiency is i itself not a contraindication to surgery At times the differentia tion between an early aortic insufficiency and pulmonic incom petency is difficult but if the signs of advanced mitral stenosi



FIGURE 34 Before and After Commissurotomy for Mitral Stenosis *(mitral area) (A) Preoperative Note the accentuated first sound* the absence of a systolic murmur and the opening snap following a faint second sound (arrow) A very loud rumbling holodiastolic murmur was present (B) Postoperative recording from the same area Note that the intensity of the first sound is about the same there is no systolic murmur the second *sound is perhaps slightly louder and it is followed* by an opening snap The previously present loud holodiastolic murmur has entirely disappeared The opening snap was heard in this case after operation but preoperatively it could not be separated by auscultation from the holodiastolic murmur Such a marked diminution of a diastolic murmur in our experience usually means a good clinical result provided no systolic murmur of note appears It does not *follow however that the clinical response will not be good if there* is no change in the murmur

are present without peripheral signs of aortic insufficiency we are inclined to consider the murmur due to pulmonic incompet ency

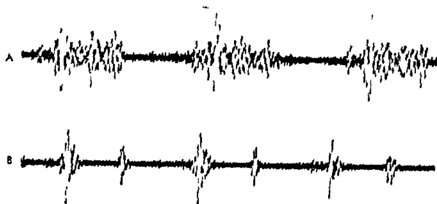


FIGURE 35 Before and After Commissurotomy for Mitral Stenosis (mitral area) (A) Preoperative recording showing a faint diastolic murmur building up to a louder presystolic murmur The first sound is followed immediately by a loud holosystolic murmur which was harsh on auscultation After the second sound an opening snap is present Because of the loud systolic murmur there was consider able doubt as to whether or not this patient should be operated on although there were good evidences of mitral stenosis such as right ventricular hypertrophy in the electrocardiogram and a large mid cardiac segment on x ray examination with little if any left ventricu lar enlargement (B) Postoperative recording from the same area about six months later Although the heart sounds are now of about the same intensity it will be noted that the systolic murmur has entirely disappeared Although there are some presystolic vibra tions and a prolonged first heart sound there is little abnormality present The possible explanations are that this systolic murmur arose from tricuspid insufficiency which disappeared after the pres sure in the right ventricle was lowered or that mitral insufficiency was actually present and as a result of the operation the mitral valve cu ps were sufficiently mobile to close The former would seem more likely The clinical improvement in this case has been marked (Courtesy of Col Thomas W Mattingly)

Generalization are difficult and each case must be individualized particularly in the presence of mitral valve calcification

In our experience mitral valve commissurotomy has a variable effect on the murmur of mitral stenosis (FIGS 34 35) The changes in murmurs may bear no relationship to clinical improvement although a marked diminution in the intensity of a diastolic murmur without the addition of a systolic murmur suggests a good prognosis

MITRAL INSUFFICIENCY

ETIOLOGIC FACTORS The most common cause is valvular deformity resulting from preceding rheumatic fever Other rare causes are bacterial endocarditis traumatic rupture of a cusp or chordae tendineae spontaneous rupture of a cusp chordae or a papillary muscle from disease and calcific disease arising from arteriosclerosis involving the cusp chordae or ring of the mitral valve

AUSCULTATORY FINDINGS A systolic murmur is heard at the apex of moderate or greater intensity (FIGS 36 37) It is frequently transmitted laterally and an accentuated pulmonic second sound is often present In most cases the murmur is blowing in quality but may be harsh or musical (FIG 38) and is of variable duration beginning immediately after the first sound In advanced mitral insufficiency the murmur is generally holosystolic The disease of the valve and the early onset of the murmur tend to diminish or *obliterate the perception of the first sound* When this murmur is present as an isolated finding the proper evaluation may be extremely difficult See page 62 for additional discussion



FIGURE 36 Systolic Murmur of Early Mitral Insufficiency (mitral area) The first sound is louder and there is a low intensity holosystolic murmur This case is interesting in that the patient was under our observation sixteen years ago at which time he was eight years of age and had a very severe rheumatic pancarditis During his long hospital stay both systolic and diastolic apical murmurs were repeatedly noted by several observers He has had no further attacks of rheumatic fever and at the present time is asymptomatic Very slight left ventricular enlargement is present This history illustrates that valvular damage may be minimal even following a very severe episode of rheumatic carditis It is interesting to note in the stethogram that there are some minor vibrations immediately after the second sound and just preceding the first sound These cannot be heard and only observation over a long period of time will determine whether or not these vibrations represent preclinical mitral stenosis

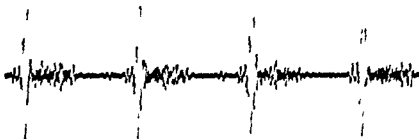


FIGURE 37 Systolic Murmur of Advanced Mitral Insufficiency (mitral area) Note the low frequency auricular vibrations (inaudible) just before the first sound which is moderately loud and is followed by a loud holosystolic murmur The second sound is of very low intensity and is not separated from the systolic murmur No deflections are present in diastole This murmur was present in a young woman who had three verified bouts of subacute bacterial endocarditis at five year intervals (Courtesy of Dr Arthur Geiger)

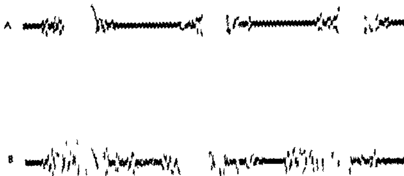


FIGURE 38 Musical Apical Systolic Murmur (A) Illustrates a long high pitched systolic murmur of relatively pure high cycle frequency with variations in intensity On auscultation this resembled the so called sea gull murmur (B) Illustrates another type of musical apical systolic murmur which on auscultation was interpreted as squeaky but which some would also call a sea gull type The cycle frequency range is still high but it is not as pure and has less variation in intensity than the murmur in A Terms of this type (sea gull etc) while frequently used for descriptive purpose have no diagnostic significance

MITRAL INCOMPETENCE

ETIOLOGIC FACTORS This condition results from enlargement of the left ventricle (FIG 23) As the ventricle enlarges the mitral ring may become dilated or the mitral cusps may become retracted by the shortening of the chordae tendineae and papillary muscles At times both factors may operate to produce the incompetence Among the causes of the left ventricular enlargement are aortic insufficiency hypertension arteriosclerosis active rheumatic fever and other types of active myocarditis certain

congenital cardiac lesions thyrotoxicosis myxedema, anemia and thiamin deficiency

AUSCULTATORY FINDINGS The murmur of mitral incompetence cannot be differentiated from the murmur of mitral insufficiency

AORTIC STENOSIS

ETIOLOGIC FACTORS The most common cause is valvular deformity resulting from preceding rheumatic fever In later life calcification may be superimposed Calcific disease arising from arteriosclerosis without preceding abnormality of the valve may also produce stenosis Congenital defects produce aortic stenosis or subaortic stenosis

AUSCULTATORY FINDINGS A loud harsh systolic murmur is heard at the right second interspace close to the sternum it is transmitted to the neck and widely over the precordium This murmur frequently appears on the stethogram in a peculiar shape commonly called diamond shaped as a result of being crescendo in its first half and decrescendo in its last half The aortic second sound is usually faint or absent and when heard distinctly usually represents transmission of an accentuated pulmonic second sound resulting from an associated mitral stenosis or mitral insufficiency (FIG 39) except in subaortic stenosis in which the second sound is unchanged Aortic stenosis may be present with only a minimal systolic murmur This is particularly true in the presence of cardiac failure FIGURE 40 illustrates a classic isolated rheumatic aortic stenosis Following aortic valve commissurotomy the second aortic sound could be heard suggesting the return of valvular function

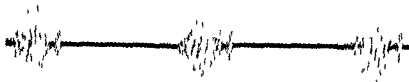


FIGURE 39 *Crescendo decrescendo Systolic Murmur of Aortic Stenosis (aortic area) The first sound cannot be separated from the murmur This type of murmur has been described from the stethographic point of view as diamond shaped as the result of crescendo in its first half and decrescendo in its last half In this case a second sound is present which is thought to be caused by transmission of an accentuated pulmonic second sound resulting from associated mitral stenosis (See FIGURE 30 for the stethogram from the mitral area in this same patient)*

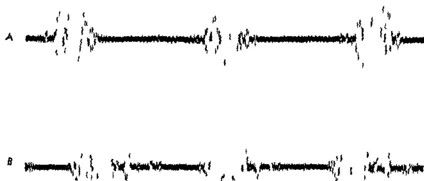


FIGURE 40 *Crescendo decrescendo Systolic Murmur of Aortic Stenosis and Return of the Aortic Second Sound after Operation (aortic area) (A) Preoperative recording showing the typical diamond shaped murmur The second sound is absent (B) Postoperative recording at about the same level of intensity The systolic murmur is essentially the same but the appearance of the second sound is believed to represent the return of some action of the aortic valve cusps A faint diastolic murmur originating in aortic insufficiency has now appeared At time of operation the mitral valve was explored and found to be normal*

AORTIC INSUFFICIENCY

ETIOLOGIC FACTORS The most common cause is valvular deformity resulting from preceding rheumatic fever Other causes are syphilis bacterial endocarditis traumatic rupture of a valve cusp rupture of a cusp due to disease calcific disease deriving from arteriosclerosis without preceding valvular abnormality dissecting aneurysm of the ascending aorta with extension into the valve area and a bicuspid aortic valve

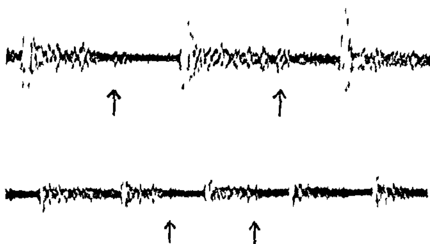


FIGURE 41 Diastolic Murmur of Aortic Insufficiency (fourth inter *space at left sternal border) There is a faint first sound (arrow)* followed by a short faint systolic murmur The second sound is loud and immediately followed by a decrescendo diastolic murmur of higher frequency On auscultation this murmur had the typical blowing decrescendo character of aortic insufficiency The upper stethogram *was recorded at 50 mm per second the lower at 25 mm per second*

AUSCULTATORY FINDINGS A high pitched decrescendo diastolic murmur beginning immediately after the second sound is heard in aortic insufficiency usually best along the left sternal

border rather than at the aortic area (FIG 41) The disease of the valve and early onset of the murmur may diminish the perception of the second sound The murmur of aortic insufficiency is frequently very faint and is easily missed (FIG 42) Transmission to the apex is not uncommon It is usually of blowing quality but may be musical when caused by a ruptured cusp (FIG 13) calcific disease or eventration of a cusp due to syphilis

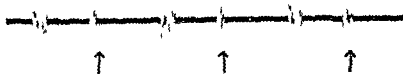


FIGURE 42 Short Diastolic Murmur of Early Aortic Insufficiency (fourth interspace at left sternal border 25 mm per second) The first sound is duplicated The second sound is louder and of short duration Immediately following this second sound is a very short decrescendo murmur (arrow) representing early aortic insufficiency Murmurs of this type are usually heard more clearly than they can be recorded stethographically because the cycle frequency (pitch) is sufficiently high to fall in an area where the human hearing mechanism has greater sensitivity (FIG 11) This situation is in contrast to such murmurs as the diastolic rumble of mitral stenosis which is easy to record but hard to hear Amplification with selective filtration is of great diagnostic aid

A procedure commonly used is to have the patient in the upright position leaning slightly forward and in deep expiration The latter is not as helpful as usually described

FIGURE 43 illustrates a diastolic murmur of aortic insufficiency with a short loud musical component Short loud musical murmurs of this type are apt to be evanescent and may appear in either systole or diastole The mechanism is unknown



FIGURE 43 Musical Diastolic Murmur of Aortic Insufficiency (fourth interspace at left sternal border) The stethogram shows a faint first sound which can be identified only with great difficulty A distinct second sound is present followed immediately by a loud musical diastolic murmur which changes abruptly to a typical decrescendo diastolic murmur This patient was a child with active rheumatic carditis On auscultation it was difficult to separate the second sound from the musical portion of the murmur which was inconstant and tended to appear and disappear while the basic murmur of aortic insufficiency was always present The mechanism of production of this short musical murmur is not clearly understood but it occurred during a period of active carditis and has disappeared with the subsidence of rheumatic activity (Courtesy of Dr Gene Stollerman)



FIGURE 44 Prolonged Diastolic Decrescendo Murmur (arrow) The patient was a 60 year old male who was hospitalized for an acute myocardial infarction and died two weeks later Review of this clinic chart did not disclose previous hypertension syphilis or the presence of this murmur At necropsy an extensive myocardial infarct was noted but no disease of the aortic valve could be demonstrated thus confirming the clinical impression that the murmur represented aortic incompetence

AORTIC INCOMPETENCE

ETIOLOGIC FACTORS The cause is dilatation of the aortic valve ring Hypertension is established as a factor In our opinion

arteriosclerosis of the ascending aorta can also cause aortic incompetence

AUSCULTATORY FINDINGS The findings are the same as those described for aortic insufficiency except that the murmur is less likely to be musical FIGURE 44 illustrates such a case

PULMONIC STENOSIS

ETIOLOGIC FACTORS Pulmonic stenosis is almost invariably a congenital lesion but a rare cause is preceding rheumatic valvulitis When *congenital it occasionally exists as an isolated* lesion and is found in two forms In the valvular type the three cusps of the valve are fused resulting in a small opening with

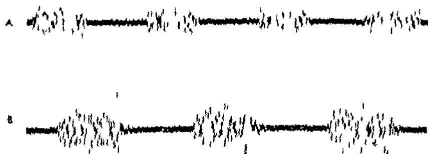


FIGURE 45 Congenital *Pulmonic* Stenosis (pulmonic area) (A) Valvular type showing the crescendo decrescendo (diamond shaped) systolic murmur *similar* to that of aortic stenosis The second sound is absent The diagnosis was *confirmed at operation* (B) *Infundibular* type The same type of systolic murmur is present in this case but the second sound is *present* The diagnosis was also verified by *operation* (*Courtesy of Dr* Edmund H Reppert)

dilatation of the pulmonary artery beyond the stenosis This dilatation is thought to be caused by a congenital weakness of the wall of the pulmonary artery The other type is the result of narrowing of the pulmonary conus two to three centimeters below a normal valve and is termed infundibular pulmonic stenosis

AUSCULTATORY FINDINGS A loud harsh systolic murmur is maximal at the pulmonic area The pulmonic second sound is greatly diminished or absent in the valvular form (FIG 45A) but only slightly if at all diminished in the infundibular type (FIG 45B) The systolic murmur is crescendo in its first half and decrescendo in the last half the term diamond shaped' has been applied as in the case of aortic stenosis

PULMONIC INSUFFICIENCY

ETIOLOGIC FACTORS The congenital form is rare and is caused by an abnormality in the number of valve cusps or by dilatation of the pulmonary artery The acquired form is also uncommon and is caused by bacterial endocarditis or a preceding rheumatic valvulitis The latter is extremely rare as is traumatic rupture of a cusp

AUSCULTATORY FINDINGS A decrescendo high pitched, blowing diastolic murmur is heard along the left sternal border, beginning immediately after the second sound It can be distinguished from the murmur of aortic insufficiency or incompetence only with difficulty When there are unequivocal signs of aortic insufficiency the differential diagnosis can be established but otherwise it may be impossible

PULMONIC INCOMPETENCE

ETIOLOGIC FACTORS Any acute or chronic process causing severe pulmonary arterial hypertension may dilate the pulmonic ring resulting in pulmonic incompetence

AUSCULTATORY FINDINGS The findings are the same as those described for pulmonic insufficiency except that the pulmonic second sound is often accentuated as a result of the pulmonary arterial hypertension and also a pulmonic systolic murmur is frequently present owing to dilatation of the pulmonary artery (FIG 46) The diastolic murmur of pulmonic incompetence is named after Graham Steell an English physician who explained its cause in 1888 [3] The following quotation illustrates the acute ness of this observer I wish to plead for the admission among the recognized auscultatory signs of disease of a murmur due to pulmonic regurgitation such regurgitation occurring independ

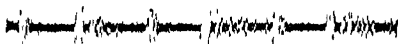


FIGURE 46 Graham Steell Murmur in a Case of Mitral Stenosis (fourth interspace at the left sternal border 25 mm per second) The stethogram shows a normal first sound and a relatively silent systolic interval followed by an accentuated second pulmonic sound After the latter there is an opening snap and then a long decrescendo higher frequency diastolic murmur of pulmonic incompetence known as a Graham Steell murmur

ently of disease or deformity of the valve and as the result of long continued excess of blood pressure in the pulmonary artery

The murmur of high pressure in the pulmonary artery is not peculiar to mitral stenosis although it is most commonly met with is a consequence of this lesion Any long continued obstruction in the pulmonary circulation may produce it

TRICUSPID STENOSIS

ETIOLOGIC FACTORS Tricuspid stenosis is uncommon The usual cause is preceding rheumatic valvulitis and it is usually associated with rheumatic disease of the mitral and/or aortic valves Tricuspid stenosis or atresia of congenital origin is extremely rare and is usually overshadowed by associated congenital defects

AUSCULTATORY FINDINGS The murmur of tricuspid stenosis is best heard at the tricuspid area and its timing may be early diastolic mid diastolic or presystolic or combinations thereof It shows considerable variation in intensity Its quality is exactly as previously described for mitral stenosis Certain observers have stated that the murmur of tricuspid stenosis frequently increases in intensity in inspiratory apnea which is not true of mitral stenosis From the practical standpoint auscultation offers little help in differentiation of these murmurs An 'opening snap of the tricuspid valve' may be heard which is so similar to the 'opening snap in mitral stenosis that differentiation is difficult When an opening snap of the tricuspid is present it is louder over the right lower precordium

TRICUSPID INSUFFICIENCY

ETIOLOGIC FACTORS This lesion is uncommon The etiologic factors are the same as those given for tricuspid stenosis In addition it may result from traumatic rupture of a cusp or chordae or spontaneous rupture of a cusp chordae or a papillary muscle owing to disease particularly bacterial endocarditis An unusual form of tricuspid insufficiency occurs in Ebstein s disease in which anomalies of the tricuspid valve and right ventricle occur

AUSCULTATORY FINDINGS A systolic murmur is best heard in the tricuspid area As with the murmur of tricuspid stenosis certain observers have stated that the systolic murmur of tricuspid insufficiency frequently becomes louder in inspiratory apnea which is not true of the murmur of mitral insufficiency Nevertheless auscultation is of little help in differentiating the murmurs of these two lesions and the murmur of tricuspid insufficiency is likewise indistinguishable from that of tricuspid incompetence

TRICUSPID INCOMPETENCE

ETIOLOGIC FACTORS This condition is common and results from enlargement of the right ventricle As the ventricle enlarges the tricuspid ring becomes dilated or the cusps of the valves may become retracted by the chordae tendineae and papillary muscles Among the causes are left ventricular failure certain forms of congenital heart disease chronic pulmonary disease and mitral stenosis

AUSCULTATORY FINDINGS The findings are the same as those previously described for tricuspid insufficiency

DILATATION OF THE ASCENDING AORTA

ETIOLOGIC FACTORS Dilatation of the ascending aorta is caused by syphilitic aortitis hypertension arteriosclerosis or trauma



FIGURE 47 Tambour Aortic Second Sound and Systolic Murmur of Dilatation of the Ascending Aorta (aortic area) The first sound is prolonged and is followed by a systolic murmur of moderate duration The second sound is very loud prolonged and of higher frequency than normal On auscultation there was a ringing or tambour quality to the second sound although this cannot be determined from the stethogram The case is one of syphilitic aortitis

AUSCULTATORY FINDINGS A systolic murmur of varying duration is heard at the aortic area and is transmitted upwards This murmur is blowing in quality and varies in intensity from very faint to moderate It differs from that of aortic stenosis since it is not ' diamond shaped In syphilitic aortitis the aortic second sound frequently has a ringing (tambour) quality and is usually accentuated (FIG 47) In hypertension accentuation of the aortic second sound is common and without change in quality In dilatation of the ascending aorta owing to arteriosclerosis only there is

no change in the second sound FIGURE 48 illustrates this murmur in a case thought to be traumatic in origin



FIGURE 48 Systolic Murmur of Dilatation of the Ascending Aorta (aortic area) This 26 year old male had been severely injured in an automobile accident nine years previously at which time several ribs were fractured and bilateral pleural effusion developed Three months after the accident cardiac auscultation was negative as was the x ray film of the chest At the time this stethogram was recorded (nine years after the accident) fluoroscopic and x ray studies showed moderate dilatation of the ascending aorta Serologic tests for syphilis were negative We consider the aortic dilatation to be traumatic in origin

DILATATION OF THE PULMONARY ARTERY

ETIOLOGIC FACTORS The chief cause is pulmonary arterial hypertension with or without secondary arteriosclerosis Increased pressure in the pulmonary artery usually arises from left ventricular failure mitral stenosis chronic pulmonary disease certain congenital cardiac lesions or acute pulmonary embolism Primary arteriolar sclerosis of the pulmonary vascular bed is a rare cause

AUSCULTATORY FINDINGS A blowing systolic murmur is heard at the pulmonic area It may be of varying duration and of very faint to moderate intensity

THE AUSTIN FLINT MURMUR

This is a presystolic murmur at the apex heard in certain cases of aortic insufficiency in the absence of mitral stenosis. It was first described in 1862 by the American physician Austin Flint[4] (1812–1886). Auscultation does not differentiate between the presystolic murmur of mitral stenosis and the Austin Flint murmur. Helpful points in diagnosis are the frequency of an opening snap of the mitral valve and of an accentuated pulmonic second sound in mitral stenosis and their lack of association with the Austin Flint murmur. The cause of this murmur is controversial and the theories that have been advanced still need experimental proof.

IMPORTANT CONGENITAL CARDIOVASCULAR LESIONS

In congenital heart disease auscultation gives a presumptive diagnosis which at times must be confirmed by angiocardiogram or cardiac catherization studies. The following section deals only with the usual auscultatory findings in common congenital lesions and is not intended as a complete diagnostic summary of congenital heart lesions.

COARCTATION OF THE AORTA

The infantile type is so named because survival beyond infancy is rare. There is a diffuse narrowing of the aorta between the origin of the left subclavian artery and the ductus arteriosus which remains patent permitting blood to pass from the pulmonary artery to the aorta below the point of narrowing. The infantile type is usually associated with other serious congenital abnormalities.

The adult type of coarctation exists in varying anatomic degrees and some individuals may live to an advanced age. It

consists of a localized constriction of the aorta beyond the origin of the left subclavian artery at or near the insertion of the ligamentum arteriosum A bicuspid aortic valve i a ociated in about

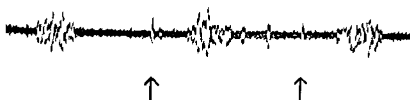


FIGURE 49 Systolic Murmur of Coarctation of the Aorta (interscapular area) There is continuous sound with marked accentuation at regular intervals which is probably the transmitted systolic murmur from the area of coarctation Low frequency low intensity vibrations which are not well visualized in the stethogram resemble a hum on auscultation They are probably due to blood circulating through dilated intercostal arteries Note two sharp spikes (arrow) appearing in the last two cycles they occur about 0 15 second prior to the murmur These sounds were automatically interjected on the peak of the R wave of the electrocardiogram and thus the interval between the spike and the murmur indicates the time necessary for blood to reach the area of coarctation after ejection from the ventricle

30 per cent of cases The adult type i e timated to be pre ent in one of every 3000 to 5000 adult

The auscultatory findings are a sy tolic murmur at the ba e and at the interscapular area often of equal inten ity although occasionally one may predominate over the other The rea on for the frequent equality of the murmurs is that the point of origin of

the murmur is about equidistant anteriorly and posteriorly The murmur may be faint in some cases anteriorly and delayed and *prolonged in duration posteriorly (FIG 49) In addition to the* congenital lesion the development of collateral arterial circulation is another factor in the interscapular murmur When a bicuspid

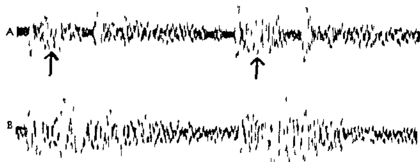


FIGURE 50 Systolic Murmur of Coarctation of the Aorta with Associated Aortic Diastolic Murmur (A) (aortic area) There is a systolic diamond shaped murmur (arrow) starting shortly after the first sound Both the first and second sounds are clearly audible thus tending to differentiate this condition from aortic stenosis Following the second sound there is a long decrescendo diastolic murmur which may arise from a bicuspid aortic valve or aortic incompetence caused by dilatation of the aortic ring as a result of the hypertension of coarctation or a combination of these two factors (B) Angle of the Left Scapula Note the loud prolonged murmur in this area which is of much longer duration than the systolic murmur seen in A The murmur reaches maximum intensity near the end of systole and continues into diastole A and B have similar time relationships and events in vertical alignment are simultaneous

aortic valve is present an aortic diastolic murmur may be associated but aortic incompetence from the accompanying hypertension may also be a cause (FIG 50) It is well to remember in connection with the interscapular murmur of coarctation of the aorta that any loud heart murmur may be transmitted to the

back When the systolic murmur is loud we have observed another phenomenon consisting of a crescendo decre cendo murmur at the apex starting appreciably after the first sound and at times carry ing through the second sound but not interfering with the latter This sequence is the result of the slight delay in the arrival of the ejection wave at the area of coarctation

ARTERIOVENOUS FISTULA OR ANASTAMOSIS

The congenital form is one of several etiologic types of arteriovenous fistula Multiple communications between the artery and vein are common in the congenital type The extremities the neck and the lungs are the u ual locations



FIGURE 51 Pulmonary Arteriovenous Fi tula (left posterior chest) Illustrated is a loud continuous murmur undulating in character

A loud continuous murmur occupying both systole and dias tole is present over the site of the fistula There is systolic accentu ation of this murmur and a thrill is generally present In the pulmonary form (FIG 51) the murmur is heard over the chest wall overlying the fistula but the intensity is affected by the location of the fistula within the chest and the size of the com munication It may be impossible to differentiate from a patent ductu arterio us by auscultation if the communication i located in the upper left pulmonary field

PATENT DUCTUS ARTERIOSUS

This lesion is actually an arteriovenous fistula between the aorta and pulmonary artery The typical murmur is loud harsh and continuous, and occupies both systole and diastole It is usually described as machinery like in character Occasionally the systolic and diastolic components are well defined but usually



FIGURE 52 Patent Ductus Arteriosus (pulmonic area) The murmur is generally described as machinery like in character It occupies both systole and diastole and is frequently undulating with systolic accentuation as in this case The upper illustration was recorded at a paper speed of 50 mm per second The lower stethogram is exactly the same but recorded at 25 mm per second

the murmur is of undulating character with systolic accentuation In rare instances the diastolic component may be absent and the systolic faint, particularly during infancy The murmur (FIG 52) is best heard at the second left intercostal space near the sternal border and if loud may be transmitted to the back After successful operation the murmur either disappears completely or there remains only a faint systolic murmur at the original site of

maximum intensity We have however seen recanalization occur if the ductus was only ligated and not divided with reappearance of the murmur

At times a venous hum may be present in the same area particularly in children It may be differentiated however in that jugular compre sion will obliterate a venous hum

ATRIAL SEPTAL DEFECT

In general an atrial septal defect is not of great clinical importance unless it is larger than two centimeters in diameter When larger the left to right shunting of blood begins to be of clinical importance Of cour e the larger the diameter of the opening between the atria the more marked the hemodynamic changes will be This is the most common of congenital defects and occurs three times more frequently in females

The auscultatory findings are probably more variable in this condition than in any other cardiac lesion The only consistent finding is an accentuated second pulmonic sound which is often split (FIC 53) A fair proportion of cases will have no cardiac murmurs while others will have systolic murmurs usually of maximal intensity at the second or third left interspace which may vary from faint to extremely loud and from blowing to harsh in quality Since the pre sure differential between the two atria is extremely small it is unlikely that these murmurs are actually produced by blood flowing through the defect but are more likely the result of the left to right shunt which throws an increa ed load on the right ventricle and results in dilatation of the pulmonary artery producing a systolic murmur The pulmonary arterial hypertension may also produce pulmonic incompetence

As in the case of many congenital defects the pathologic changes are not single but multiple and frequently some degree

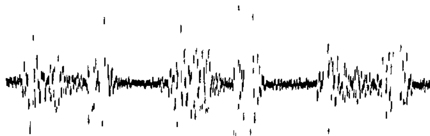


FIGURE 53 Atrial Septal Defect (pulmonic area) The systolic murmur varies from cycle to cycle and the second sound is accentuated and duplicated Auscultatory findings in this defect vary widely from case to case

of pulmonic stenosis may occur simultaneously with atrial defects Since the murmurs of both may be quite similar the final diagnosis requires cardiac catheterization although a harsh loud, diamond shaped murmur is suggestive of the presence of pulmonic stenosis

VENTRICULAR SEPTAL DEFECT

The common defect seen in adults is usually small in size and located *near the basal part of the septum* It is characterized by a harsh systolic murmur varying in intensity from loud to very loud which is heard best in the third or fourth left intercostal space along the sternal border (FIG 54) As with all systolic murmurs produced by flow through a small orifice under high pressure this may also have a crescendo decrescendo or 'diamond shaped character This is frequently called a Roger murmur after the French physician Henri Louis Roger who first described this entity * The heart sounds are usually normal but

* This cardiac anomaly has no *objective symptom which the eye can* recognize it belongs almost exclusively to the field of auscultation After



FIGURE 54 Ventricular Septal Defect (third interspace at left sternal border) This systolic murmur sounded harsh Clinically it was asso ciated with a thrill An extra sound of unknown cause in early diastole is present In the stethogram this resembles an opening snap but on auscultation it was interpreted as the second heart sound Actually the second sound is incorporated with the systolic murmur and thus was not recognized on auscultation

the second pulmonic sound may frequently be accentuated as a result of pulmonary hypertension particularly when the defect is large

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having auscultated thousand of children during forty years of special tudies and thanks to stetho copic examinations unceasingly repeated I have been able with the control of pathologic anatomy to separate this anomaly of the heart from other malformations or diseases to establish its distinct clinical existence and to make of the murmur which characterizes it a pathognomonic sign

It was in the amphitheatre of L Hopital De Enfants about 1861 that I saw the light and I discovered the reason for the e ob curities and apparent contradictions In a young boy of twelve dead of a comminuted fracture I found at necrop y a defect of tructure which con i ted of a failure of occlu sion of the interventricular partition in its upper part without concomitant stenosi of the pulmonary artery in spite of the free mixture of the two blo ds which had resulted neither the skin nor the ti ues had been blue during life It goes without saying that this malformati n had been completely unrecognized by a very pardonable oversight on a urgical ervice neglecting to listen to the heart (Our tran lation)

THE TETRALOGY OF FALLOT

This condition is characterized by (1) dextroposition of the aorta, (2) ventricular septal defect (3) pulmonic stenosis (4) right ventricular hypertrophy The loud harsh systolic murmur heard best in the second or third left intercostal space, along the sternal border is the result of pulmonic stenosis and the ventricular septal defect *One cannot distinguish the origin of the murmur* The pulmonic second sound is variable depending upon whether or not the stenosis is valvular or infundibular

EISENMENGER S COMPLEX

*This congenital abnormality is often confused with the tetral*ogy of Fallot However, pulmonic stenosis is absent in Eisenmenger s complex and pulmonary hypertension is invariably pres*ent, resulting in a loud second pulmonic sound Occasionally, with* the marked increase in pulmonic pressure the pulmonic ring may dilate giving rise to a diastolic murmur of pulmonic incompetence A loud harsh systolic murmur at the third or fourth left inter space is the result of the ventricular septal defect

LUTEMBACHER S SYNDROME

Mitral stenosis of either the congenital or acquired type combined with an auricular *septal defect constitutes Lutembacher s* syndrome The auscultatory findings are primarily those of mitral stenosis

AUSCULTATORY FINDINGS AS RELATED TO ETIOLOGIC FACTORS

Certain abnormalities often thought to be as ociated with heart di ease, such as nonpathologic murmurs arrhythmias extra

cardiac sounds and rubs will be omitted in this discussion but can be found elsewhere in this book

Heart disease caused by *rheumatic fever* may be classified as active or inactive rheumatic heart disease The former means that the process is active while the latter refers to the residual damage caused by this process It is not always possible to be certain of the presence or absence of rheumatic activity of the chronic type and of course both active and inactive rheumatic heart disease may be present in the same patient when a recurrence of activity occurs

Acute active rheumatic heart disease is best discussed on the basis of a first attack of rheumatic fever It is established that mitral incompetence with an apical systolic murmur can result from enlargement of the left ventricle owing to rheumatic myocarditis but the development of mitral insufficiency mitral stenosis or aortic insufficiency during this phase seems unlikely to us However mitral valvulitis in this stage of the disease occasionally is associated with a low pitched apical mid diastolic murmur and acute active aortic valvulitis may produce a high pitched aortic diastolic murmur All of these murmurs may prove to be transitory but in the presence of chronic active valvulitis permanent valvular deformity usually develops (FIG 36) Involvement of the pulmonic and tricuspid valves is unusual in rheumatic heart disease

In *inactive rheumatic heart disease* varying degrees of mitral stenosis and insufficiency are often found together aortic stenosis and insufficiency are frequently associated and varying combinations of aortic and mitral valve lesions exist in many cases

Arteriosclerotic heart disease often has a systolic murmur at the aortic area caused by dilatation of the ascending aorta or

an apical systolic murmur of mitral incompetence or both Less commonly arteriosclerosis with calcification produces mitral insufficiency, aortic stenosis or aortic insufficiency Arteriosclerosis of the aorta may occasionally produce dilatation of the aortic valve ring resulting in aortic incompetence

Hypertensive heart disease may show mitral incompetence from dilatation of the left ventricle aortic incompetence from *dilatation of the aortic valve ring* or *an aortic systolic murmur* arising from dilatation of the ascending aorta beyond the ring which may be accompanied by an accentuated aortic second sound

A discussion will be found earlier in this chapter regarding *congenital cardiovascular lesions* In general it may be said that loud harsh systolic murmurs heard best along the left sternal border are more indicative of a congenital lesion than an acquired one

When *syphilis* is the etiologic factor and the disease is limited to the ascending aorta one may hear a very faint to moderate blowing aortic systolic murmur which is transmitted upwards The aortic second sound may but does not always have a ringing or tambour quality This murmur derives from dilatation of the ascending aorta the alteration of the second sound is thought to result from changes in the wall of the aorta, but occasionally this type of sound can be heard in hypertensive heart disease If the syphilitic process extends downwards into the aortic valve the auscultatory findings are then in addition those of aortic insufficiency caused by valvular deformity In advanced cases with enlargement of the left ventricle mitral incompetence may occur An Austin Flint murmur is occasionally heard (page 94)

In *hyperthyroidism* the first sound at the apex is accentuated and is often sharp and napping Nonpathologic systolic murmurs

based on the associated tachycardia may be heard at the base and apex However an apical systolic murmur of mitral incompetence may be present originating from left ventricular dilatation which may develop in hyperthyroidism of long duration If there is underlying heart disease in addition to the hyperthyroidism the tendency to dilatation is increased and the associated heart disease obscured

In *hypothyroidism* affecting the heart the auscultatory findings are bradycardia and faintness of heart sounds The latter may in part be caused by the presence of a pericardial effusion

Heart disease arising from *chronic pulmonary arterial hypertension* results from diffuse disease of the lung parenchyma particularly pulmonary emphysema and fibrosis or disease of the pulmonary arterial system such as pulmonary arteriolar sclerosis or extrinsic pressure upon the pulmonary artery This excludes those cases in which increased pulmonary arterial pressure is based upon mitral stenosis left ventricular failure acute cor pulmonale or certain types of congenital cardiovascular defects and so represents true chronic pulmonary heart disease The cardiac auscultatory findings preceding the eventual failure of the right ventricle consist of a Graham Steell murmur (page 89) an accentuated pulmonic second sound and occasionally a blowing pulmonic systolic murmur deriving from dilatation of the pulmonary artery To the above is usually added the systolic murmur of tricuspid incompetence on the development of right ventricular failure

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